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ANNALS
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XLIV

THE ORIGIN OF LARYNGOLOGY

D. BRYSON DELAVAN, M.D.

NEW YORK

Those of the present generation who are interested in the science of laryngology and are now reaping the many benefits which have arisen from it, must never cease to remember the great men to whom we primarily owe its origin. Surely we are under mighty obligations to them.

The history of laryngology properly begins with the discovery of the effective means of demonstrating the interior of the larynx. Prior to the coming of the laryngoscope, however, many diseases of the throat had been studied and considerable written about them. Indeed, as long ago as the year 1650, and occasionally later, there were a few publications which refer to it. In the time of the late Horace Green, the subject embraced a large number of conditions of the upper air passages which demanded attention and which, in many cases, received treatment which we today could hardly countenance. In 1846, Horace Green, who had made an especial study of the whole subject, published his treatise on "Bronchitis." In this, a veritable pioneer, he advocated the use of topical applications to the interior of the larynx, and in cases of urgent dyspnea, of the admission of inspired air to the lungs by means of catheterization of the larynx. Dyspnea being overcome by this method of catheterization, the necessity for tracheotomy was avoided. Green was successful in applying to the larynx solutions in which a slender frag-

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ment of sponge had been bathed. The sponge was fastened to the end of a small whale-bone rod, one extremity of which was bent at such an angle that the sponge could be inserted into the larynx and the medicine applied to its surfaces. A weak solution of nitrate of silver proved to be his most effective medicament. The great discovery of Dr. Green, however, was that of the possibility of the tolerance of the larynx to the temporary presence of a foreign body.

Such was the progress then made, and the treatment of the throat and larynx was developed to an extent that in later years Clinton Wagner truthfully said, "If the laryngoscope had never been discovered, the methods of Horace Green would in themselves have founded a department of medicine." He was universally acclaimed the "Father of Laryngology."

In 1848 Gurdon Buck, the distinguished general surgeon of his time, who had already gained great credit for skill in tracheotomy, and who was the first to perform laryngofissure for the removal of an intralaryngeal growth, published his first epoch-making paper upon "Edematous Laryngitis and its Treatment by Scarification." This was the first step toward the surgical treatment of the interior of the larynx. The work of both of these two great men was enthusiastically proclaimed by those who understood its value, both here and abroad, and Dr. Gurdon Buck received the well-earned title of "Father of Laryngeal Surgery."

Meanwhile, notwithstanding the progress made in the treatment of its diseases and a somewhat better understanding of its physiology, the interior of the larynx had been concealed from view. Manuel Garcia, of Spanish birth, a famous teacher of singing, devised an instrument by which the vocal cords could be clearly demonstrated to the eye, thus succeeding where several former attempts had failed (1858).

Two years later the laryngoscope of Garcia was perfected by Czermak of Pesth and was introduced into Europe. Its value was at once appreciated by Horace Green, who secured the fist example of the apparatus brought to this country.

Professor Ludwig Türck of Vienna at once adopted it and soon became the original instructor of the foremost of the followers of the art. Following the brilliant leadership of Horace Green, who had long been an acknowledged authority upon the subject of diseases of the throat, the study of them after the discovery of the laryngoscope was conducted in no uncertain way, both here and in

Europe. Vienna became the great center of instruction, while Paris also developed several great authorities.

A master of the art was Morell Mackenzie of London, whose book on the one hand and whose especial hospital in London were the means of educating a wide circle of advanced students, many of whom became acknowledged leaders. Among these was Clinton Wagner of Baltimore who, having thoroughly prepared himself by a long residence at the London Throat Hospital of Morell Mackenzie and by other superior means abroad, established in the early '70's a special throat hospital in New York City where a number of the foremost specialists of the time were educated.

In 1873, Clinton Wagner, appreciating the advantages to be derived from a society for the consideration of laryngological work, called a meeting of several who had studied the art in Vienna and elsewhere, the best who held positions in New York City, and organized what was known as the "Laryngological Society of New York." This was the first society devoted exclusively to laryngology and rhinology established either in this country or in Europe. Five years after, in the city of Buffalo, N. Y., in 1878, the American Laryngological Association was organized. Not until ten years later, in 1888, was a similar society organized in Europe, when Morell Mackenzie established the Laryngological Society of England.

In 1879, Dr. Luis F. Sass presented before the New York Academy of Medicine an entirely new method for the cleansing of the upper air passages and for the application to them of various therapeutic agents by means of the atomized spray. This method at once superseded the older forms of application to the throat and nose, for it was less trying to the patient, far more thorough and absolutely aseptic.

One of the most valuable contributions ever made to laryngology was that of Joseph O'Dwyer of New York who, in 1880, demonstrated that a foreign body could be tolerated by the larynx indefinitely. O'Dwyer devised and perfected the method known as intubation. While the use of the sponge probang and later the brush for making applications to the interior of the larynx had been universally adopted, full credit for intubation is universally conceded to O'Dwyer, who first demonstrated the principle of the continued tolerance of the larynx to the presence of a foreign body. O'Dwyer's personal character, as well as his achievements as a pathologist and inventor, ranked him among the great men of his time. No one improved upon his intubation tube, whereby the operation of

tracheotomy, formerly often demanded, was unnecessary. He foresaw other conditions for which his tube, or a modification of it, might be useful, since it offered a rational method for the dilatation of chronic stricture of the larynx. Still later, he foresaw the value of his idea for the expulsion of foreign bodies from the trachea and bronchi. For this he devised a tube of the largest possible calibre and thinnest walls. This tube was inserted into the larynx, the patient was placed upon his back and, to a certain extent, inverted, and the foreign body extruded by the coughing and other proper efforts of the patient himself—a means of saving life in the easiest and most direct manner. What is of greatest importance, however, it was the forerunner of the now worldwide method of bronchoscopy, the origin of which is claimed by many. Its adaptation to various conditions of the upper air passages in addition to the extraction of foreign bodies from the lung has been effected. Although much has been claimed as to its origin, its fundamental principle is due to O'Dwyer, to whom all credit should be given as its actual discoverer.

Since its early days the science of laryngology has developed to such an extent that long ago it became a study demanding serious effort on the part of those who had the ability to appreciate what it had already accomplished and the foresight to realize the necessity for the investigation of the new problems which are always confronting it. Some of the most vital of them have not yet been solved.

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XLV

INFECTION AS A CAUSE OF FIBROSIS OF THE ESOPHAGUS

HARRIS P. MOSHER, M.D.

MARBLEHEAD, MASS.

Early in my dealing with so-called cardiospasm of the esophagus, I became convinced that spasm played but a minor part in cases as they came to our hospital. Under the fluoroscope there are generally no signs of peristalsis, therefore, no signs of spasm. In the last ten years, Dr. Macmillan and I cannot have seen more than eight or ten cases in which spasm was a prominent feature. Examination with the esophagoscope convinced me that many of these were really cases of stricture of the terminal portion of the esophagus. Often the stricture consisted of only a slight gluing of the vertical folds which are so prominent in the terminal portion of the esophagus. At first I was at a loss for a cause of these strictures. Then I began to find in autopsy specimens, especially in specimens where the patient had gall-bladder infection, that the terminal portion of the esophagus was infected and fibrosed. One striking instance of this is shown in Figs. 1 and 2. I have not had many autopsy specimens from patients who died of cardiospasm. My friends, however, in different parts of the country have contributed a few so that, including a case or two of my own, I have been able to examine the esophagus in some five cases. In all of the specimens there was a fibrosis of the terminal portion of the esophagus associated with a disorganization of the musculature (Fig. 3). The esophagus above the terminal portion, that is, in a long-standing case, showed fibrosis also but to a less extent. This made me feel that the essential lesion in cardiospasm is a fibrosis of the terminal portion of the esophagus due to infection from neighboring organs, especially the gall-bladder and the liver.

The frequent finding of webs at the upper end of the esophagus and below the cricoid cartilage not associated with cervical exostosis left me in the dark as to their cause (Fig. 4). Then came the finding that the esophagus can be infected in acute disease as well as in chronic, for example, in pneumonia. A further finding was that in chronic infection of the esophagus and in degenerative diseases

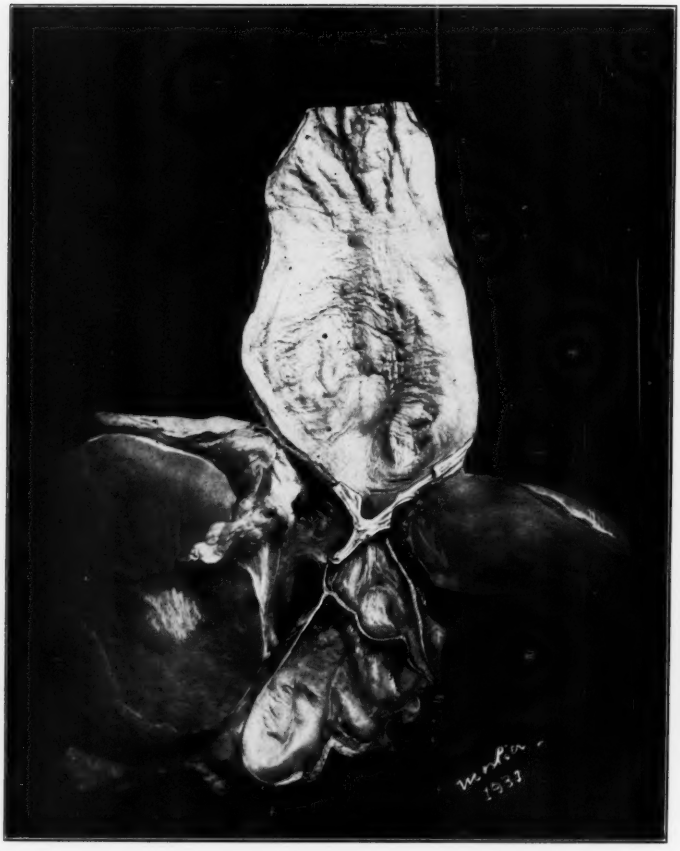


Fig. 1. Front view. The patient from whom this specimen was obtained was treated by the writer in the early days of his work on the esophagus. It was a typical case of what was then called cardiospasm. There was a double bend at the terminal portion of the esophagus. The fibrotic area was dilated with difficulty. The patient refused further treatment. The amount of dliatation obtained allowed the patient to live on soft solids. At the end of ten years she died of suffocation from the overflow of liquid from her esophagus.

The specimen showed a small scarred liver, and a chronically infected gall bladder. There was a large band-like adhesion on the front of the terminal portion of the esophagus, and multiple adhesions posteriorly.

The microscopic section of the constricted terminal portion of the esophagus showed that the musculature was almost completely replaced by fibrous tissue.

This is an extreme case of fibrosis of the terminal portion of the esophagus from infection from neighboring organs. (See Fig. 2.)

Fig. 2. Rear view of the specimen shown in Fig. 1.

like arteriosclerosis, there is marked increase in the fibrous tissue, in many cases amounting to an obliteration of parts of the circular or longitudinal muscular layers. It became evident, therefore, that the esophagus could be infected not only from without and from the blood stream, but infected from within, probably by way of the glands. It is extremely common in chronic infection to find an increase of the subepithelial connective tissue of the esophagus. These findings, plus the fact that periesophageal hemorrhage and hemorrhage into the musculature of the esophagus occur, give, I feel, abundant cause for webs or strictures of the esophagus at any part of its course. It becomes unnecessary, therefore, to rely on spasm, espe-

Fig. 3. Male, 70. Fibrosis of the terminal portion of the esophagus. Microphotograph of a section taken from the terminal portion of the esophagus. Mallory stain. The esophagus is completely disorganized. What is left of the circular layer is mostly fibrous tissue. There is a great increase of periesophageal connective tissue. Some muscle bundles of the crura show in this. The stain shows that they are beginning to be replaced by fibrous tissue.

cially at the lower end of the esophagus, as the chief cause of esophageal obstruction.

MATERIAL

The main material for the paper consists of one hundred autopsy specimens of the esophagus. The macroscopic and microscopic findings are the basis of the paper.

In the study it was first proved that fibrosis of the terminal portion of the esophagus can be due to infection from without, namely from the gall bladder or the liver. Then it was found that infection could proceed from within outward, starting in the sub-

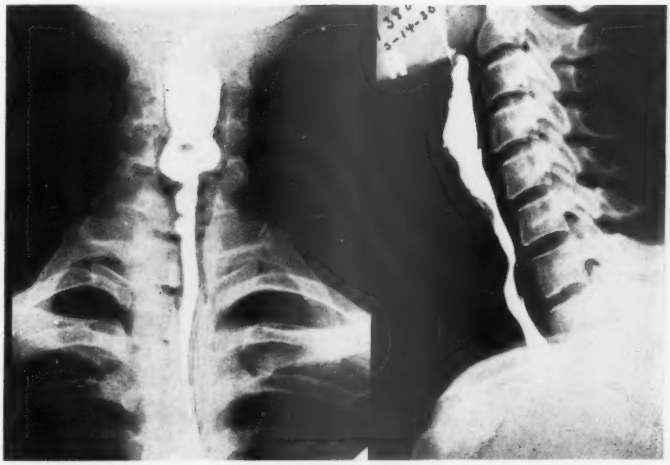


Fig. 4. Multiple fibrous strictures beginning at the lower border of the cricoid cartilage. Such strictures have been found to be even more common than fibrous strictures at the lower end of the esophagus. The writer believes that the infection which causes them originates in the pharynx or the apices of the lungs.

epithelial connective tissue and extending through the muscular layers. When it pursues this course, the *muscularis mucosa* is involved first, and then the circular and the longitudinal muscular layers in turn. When only one muscular layer is involved it is usually the circular.

THE CONNECTIVE TISSUE OF THE ESOPHAGUS

The muscular layers of the esophagus have their natural connective tissue sheaths, and in this there is much elastic tissue which is more abundant at the lower end. The muscle bundles also are separated by connective tissue. There is much periesophageal connective tissue which acts as a bed for the esophagus. Our present interest in these connective tissue bundles or sheaths is that the esophageal connective tissue is the first to fibrose as the result of infection (Figs. 5 and 6).

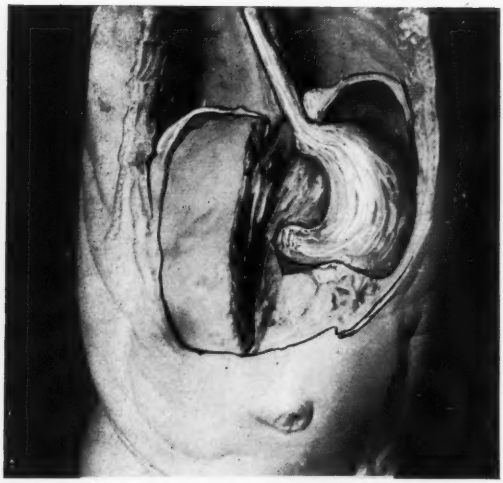


Fig. 5. Baby dead at birth dissected to show the relation of the lesser omentum to the stomach and to the lower end of the esophagus. Notice the thickening of the left crural angle. The writer believes that the angle between the left crus and the esophagus is the point of election for thickening of the connective tissue about the esophagus, the thickening being due to infection of the glands in this locality. The esophagus is surrounded by a very abundant connective tissue sheath. This is continuous, without break, with the lesser omentum. The connective tissue is more abundant between the left crus and the esophagus. Many glands are placed in the sheath. In one specimen of the esophagus of a baby dead at birth, there was a great enlargement of the glands at the bifurcation of the trachea. Both trachea and esophagus were surrounded by glands at this point, and a tubular sheath of glands followed the esophagus to the esophageal opening in the diaphragm. Between the crura and the esophagus, especially between the left crus and the esophagus, there was a nest of glands suggesting a plexus similar to the one at the bifurcation of the trachea.

CHANGES DUE TO INFECTION

Acute infection arising within the esophagus occurs either as superficial ulceration or as edema of the submucosa and infiltration of the connective tissue. At least three instances are recorded in which the submucosa is fully as thick as the two muscular layers together, the muscular layers not being involved (Figs. 7 and 8).

Chronic infection of the esophagus is shown by a generalized infiltration with small round lymphocytes and plasma cells and by an increase in the subepithelial connective tissue, or by an increase

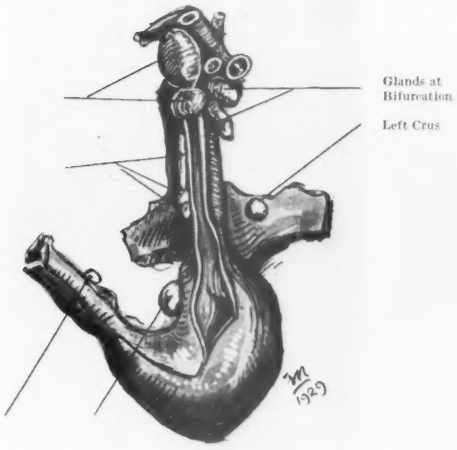


Fig. 6. The esophagus and stomach of a baby dead at birth. All markers not labeled indicate glands. The purpose of the illustration is to show the position of the lymphatic glands about the esophagus. The glandular enlargement in this specimen is typical of that found in tuberculosis. There is a large mass of glands at the bifurcation of the trachea which surrounds both the trachea and the esophagus. There is a glandular chain on both sides of the esophagus down to the diaphragm. This is more marked on the right. There is a good-sized gland between the left crus and the esophagus. Other specimens show that this gland is very constant. Below the diaphragm on the right and at the top of the lesser curvature of the stomach there is another large gland. This also is generally found. The illustration shows a small gland at the pylorus of the stomach.

in the connective tissue throughout the esophagus and in the periesophageal connective tissue bed. The lymphocytes often form a ribbon-like band under the epithelium. They collect in the glands especially about the ducts. In one instance, the collections about ducts resembled solitary follicles. In chronic infection, the number of solitary follicles under the epithelium greatly increases.

The glands, the writer feels, play a large part in infecting the esophagus from within, especially the superficial glands as these are almost beneath the epithelium. It has been observed by others that in the upper part of the esophagus the superficial glands are often

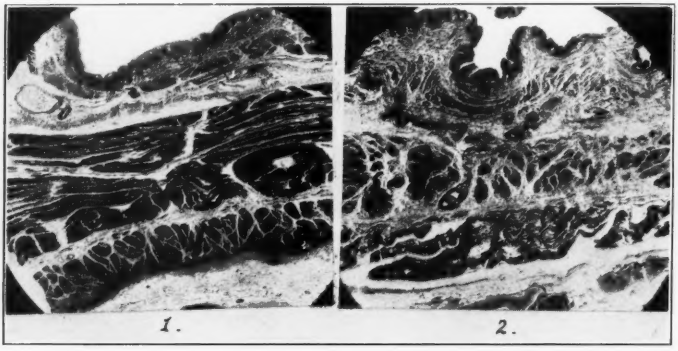


Fig. 7. The microphotograph is from a section of the esophagus taken at the middle and so well above the terminal portion. The second section is taken from the tubular, narrowed portion. In the first section there is no increase of fibrous tissue. In the second section, which is slightly thicker than the first, there is a great increase of fibrous tissue, especially below the epithelium and between the muscle bundles of the circular layer. The circular layer in the second specimen is only half as thick as in the first specimen. Normally it should be of equal thickness in both places. The muscle bundles both of the muscularis mucosa and of the circular layer are widely scattered by the increase of fibrous tissue. The first section is cut transverse to the long axis of the esophagus, the second parallel to it.

found to be eroded, making shallow ulcers. It is not uncommon also to find large solitary follicles over which the epithelium is lacking, looking as if they had pushed the epithelium off as they enlarged (Fig. 9).

The surface epithelium and the epithelium of the gland ducts seem to have a great attraction for the small lymphocytes. The cells of the glands attract them also, but to a less extent. When the lymphocytes are numerous under the surface epithelium, they swarm through the basement membrane at various points, and are seen in numbers in the epithelium. At the places where they go through, the basement membrane is broken up, and the lower cells of the epithelium take on a confused arrangement strikingly like the beginning of malignant disease.

The epithelium of the esophagus, since there are about 16 inches of it and because it can be followed so easily, would seem to be an ideal structure in which to trace the beginning cellular changes in malignant disease.

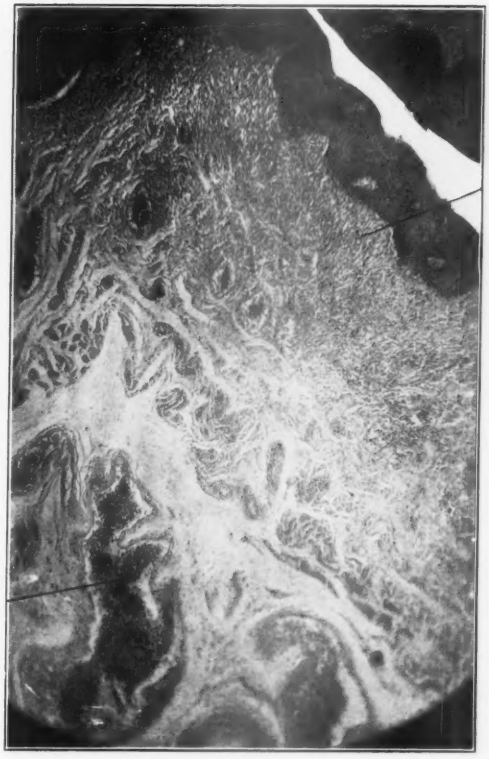


Fig. 8. The section shows great thickening of the subepithelial connective tissue. (See marker.)

Now to substantiate the above statements by the findings in various specimens.

The Esophagus at Birth. I have specimens showing that the esophagus can be markedly infected at birth. I have found one case of ulcer at the lower end, also two specimens showing a backward bend of the terminal portion. Such a bend plays a prominent part in cardiospasm.

At birth it is not uncommon to find hemorrhage into the esophagus or marked perioesophageal hemorrhage. The pediatricians maintain that this is due to asphyxia occurring during delivery. I have

Fig. 9. Microphotograph of a section of the esophagus taken at the level of a perforation but at a spot where the esophagus appeared normal. The section shows a great increase of connective tissue under the epithelium and outside the longitudinal layer. In the longitudinal layer itself there is also a great increase of fibrous tissue. In one place it has nearly replaced a group of muscle bundles.

a feeling, however, that it may be associated also with sepsis (Figs. 10, 11 and 12).

Narrowing of the cardia at birth. I have three specimens showing marked narrowing of the cardiac end of the esophagus at birth with dilatation of the esophagus above the strictured area and narrowing of the pylorus as well. Congenital narrowing of the pylorus alone has long been known. If the operative procedure for the treatment of congenital stenosis of the pylorus should prove unsuccessful the surgeon has a ready alibi in the possible presence of a narrowing at the cardia also. The specimens just enumerated show that the esophagus can be in marked trouble before birth.

Infection of the Esophagus in Cirrhosis of the Liver. It is old knowledge that the esophageal vessels are greatly dilated in cases of cirrhosis of the liver due to back pressure. It has also long been

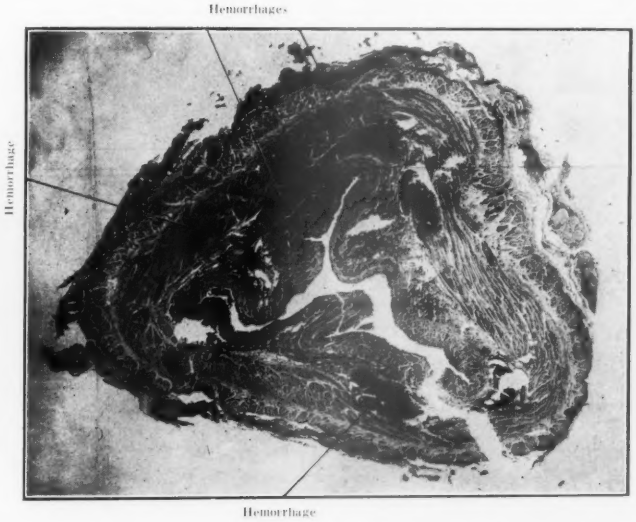


Fig. 10. Baby died at birth from intracranial hemorrhage. The section of the esophagus shows many large hemorrhages.

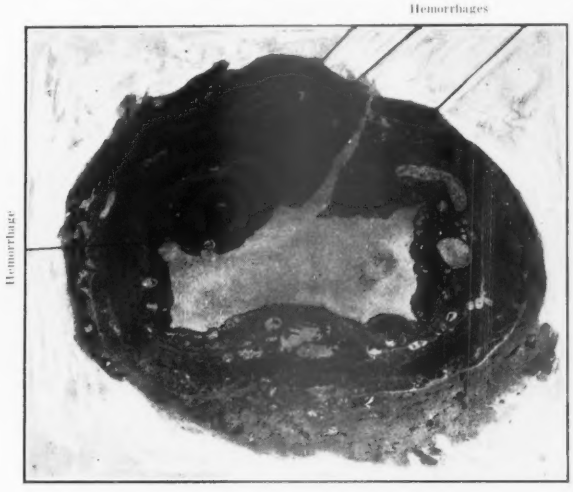


Fig. 11. Premature baby—8 months—dead at birth. Mother died of eclampsia. The section shows numerous hemorrhages.

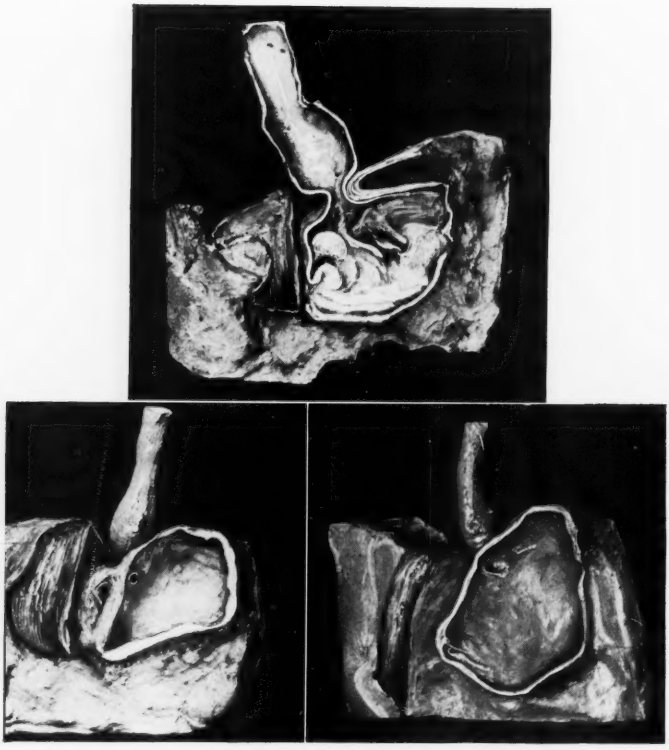


Fig. 12. Retouched photographs of three babies dead at birth. The upper figure is from a case of congenital stenosis of the pylorus associated with stenosis of the cardiac end of the esophagus. The lower third of the esophagus is much dilated. The esophagus shows three small pouches. The right half of the stomach from the pylorus to the cardia is a mass of large welts. The cardiac end of the esophagus is a fibrous ring. Histological examination shows that the muscles of the thickened part of the stomach are greatly hypertrophied. There is also a great increase of fibrous tissue. The same condition is present at the cardiac end of the esophagus and there are many glands between the crura and the esophagus.

The lower figures also show a narrowing of the pylorus associated with a narrowing of the cardiac end of the esophagus. In each of the two, the lower third of the esophagus is dilated. The musculature of the stomach of the lower two babies was not hypertrophied.

These three babies with narrowing of the pylorus associated with narrowing of the cardiac end of the esophagus were found in the examination of sixty-five babies dead at birth.

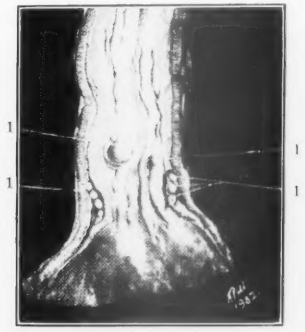


Fig. 13. Atrophic cirrhosis of the liver. The picture shows, *1*, cysts projecting into the esophageal lumen and cysts under the mucous membrane. In chronic infection of the esophagus infected glands and cystic gland ducts are common.

known that the dilated vessels at times bleed, enough even to cause death. The rupture, even in fatal cases, may be so small that it cannot be located at autopsy.

The specimen in Fig. 13 taken from a case of atrophic cirrhosis of the liver, was 10 inches long. The upper two inches were soft. The remainder was hard, almost like cartilage. At the lower end there were a number of enlarged glands. One measured fully 1 cm. in each diameter. On section, the whole of the firm part of the esophagus was seen to be studded with cystic glands.

On microscopic section, the gland ducts were found to be greatly dilated. Occasionally one was found to be filled with pus. In places fibrosis of the circular muscular layer occurred.

In this case, there were dilated and infected gland ducts and fibrosis of the muscular walls of the esophagus. The cystic ducts plus the fibrosis of the muscular layer gave the esophagus its cartilaginous feel (Figs. 14, 15, 16).

In another specimen from a case of cirrhosis of the liver, the microscopic sections showed veins which were dilated almost to the bursting point, especially at the cardia. At the cardia there was a gland. The muscles of the esophagus, where the gland was attached, were thin and mostly replaced by fibrous tissue. There were areas of fibrosis about the enlarged superficial vessels. There were not many enlarged gland ducts.

Fig. 14. Atrophic cirrhosis of the liver. The illustration is a cross-section of the esophagus shown in Fig. 13. The esophagus is riddled with cysts.

INFECTION OF THE ESOPHAGUS FROM WITHOUT

The following two cases illustrate fibrosis of the terminal portion of the esophagus due to infection from without.

CASE 1.—F. O. A., a white man, aged 63, had had a peptic ulcer 15 years previously, for which posterior gastrojejunostomy was performed. He began to show signs of obstruction, and two weeks prior to death was again operated on, cecostomy being performed. The symptoms and signs of peritonitis and further obstruction then developed, and he died.

Autopsy Observations.—Autopsy was performed ten hours after death. It showed the old posterior gastrojejunostomy (the peptic ulcer itself was not found), with ulceration, hemorrhage and perforation with obstruction. The following additional changes were noted: the recent cecostomy with a localized abscess, multiple intestinal adhesions, mitral, aortic and myocardial fibrosis, coronary sclerosis, healed apical tuberculosis, healed perihepatitis, chronic splenitis, arteriosclerosis with nephritis and a diverticulum of the urinary bladder. The anterior surface of the pancreas was adherent to the posterior wall of the stomach, partially sealing the perforating ulcer.

Macroscopic Examination.—The lower end of the esophagus at the angle where the esophagus joins the stomach was markedly thickened and fibrosed. The cardiac opening was narrow and puckered into a fibrous ring. For about four

Fig. 15. Cirrhosis of the Liver. The vessels are dilated especially the veins. The gland ducts are much enlarged and are filled with infected material.

inches above this, the esophageal musculature was thickened. Above the thickened portion, the esophagus was only half as thick, that is, about 2 mm.

The fibrosis of the cardia was probably due to infection from the previous ulcer of the stomach.

Microscopic Examination.—The microscopic examination showed extreme fibrosis at the junction of the esophagus and stomach.

The slide showed dilated and infected glands, and in the mucosa of the esophagus in one place there was a definite abscess with marked infection in and about it. This, of course, is a marked case. Lesser degrees, in fact, all degrees, of fibrosis occur.

CASE 2.—A woman had fibrosis of the terminal portion of the esophagus for at least 12 years. Once during that period I managed to dilate the narrowing sufficiently for her to get along in some comfort. She refused a second dilatation. She promised me her esophagus when she died, and she kept her word, or rather her relatives did.

Fig. 16. The esophagus (adult) from which this section was taken showed a small dimple half the size of the head of an ordinary pin. Opposite this, glued to the outside of the esophagus, there was a gland. The section shows what the writer has never before been fortunate enough to get a specimen of—a beginning traction pouch of the esophagus.

The lower half of the esophagus was dilated so that it looked like a second stomach. (See Figs. 1 and 2.)

Microscopic Examination.—The microscopic sections showed that the part of the esophagus in the crural canal had little remaining muscular tissue, most of the tissue being fibrous. The liver was small and scarred. The gall-bladder was thickened and infected, and the terminal portion of the esophagus was surrounded by stout adhesions which sprang from the sheath of the gall-bladder. The disintegration of the muscular tissue extended to the fundus of the stomach.

As chronic infection and fibrosis of the terminal portion of the esophagus from infection from without is an old subject on which I have written a good deal, I shall stop with these two rather extreme examples and go on to a newer subject, namely, involvement of the esophagus in acute disease.

INVOLVEMENT OF THE ESOPHAGUS IN ACUTE INFECTION

In connection with acute infection of the esophagus, it is not uncommon to have an exudate form over the site of the infected area. A case of general peritonitis is a case in point.

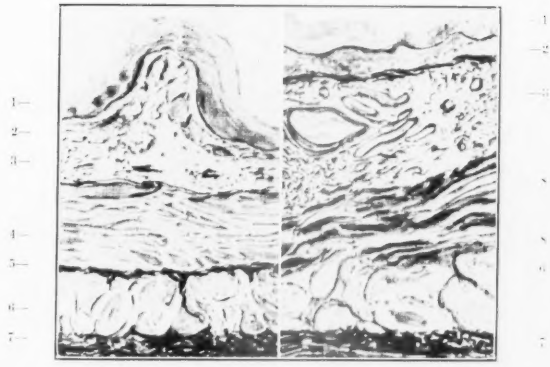


Fig. 17. The first photograph shows a specimen from a normal esophagus. The specimen shows the normal amount of connective tissue. The largest amount is between the muscularis mucosa and the circular muscular layer. If the muscularis mucosa is absent, the connective tissue is subepithelial. In this specimen there is a small amount of connective tissue between the epithelium and between it and the muscularis mucosa. There is also a small amount between the circular and longitudinal muscular layers. The greatest amount is between the muscularis mucosa and the circular muscular layer. Outside the esophagus, there is normally much connective tissue. This is not shown in the section.

Second photograph. Autopsy diagnosis: chronic cholecystitis and cholelithiasis and icterus. The specimen of the esophagus was 11 inches long and bile-stained throughout. In the lower third, there were a few cysts in the vertical rugae. The photomicrograph was taken from a section 1 inch above the cardia. The section shows a great increase of connective tissue. The subepithelial connective tissue is increased in amount and is edematous. The subepithelial vessels are enlarged. The circular muscular layer is almost obliterated by fibrous tissue. Other sections show enlarged and infected gland ducts. All the sections show chronic infection. This specimen is a striking example of fibrosis of the terminal portion of the esophagus associated with chronic infection of the gall bladder.

1, Epithelium; 2, muscular mucosa; 3, superficial layer of connective tissue; 4, circular muscular layer; 5, connective tissue; 6, longitudinal muscular layer; 7, periesophageal connective tissue; 8, fibrous tissue nearly replacing circular muscular layer.

The esophagus in this case is spattered with numerous flakes of exudate which cover ulcerated areas. The presence of the exudate over the ulcerations would, in the healing of the ulcers, make the chances of the formation of a web or a stricture much greater.

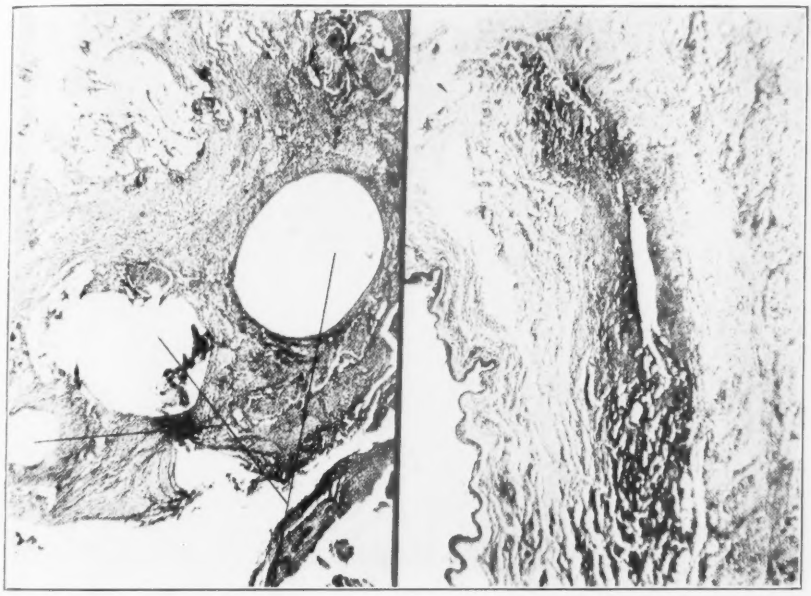
In the resting state of the esophagus the vertical folds of the terminal portion are practically in apposition, and it requires but a slight amount of infection to glue them together and to start obstruction.

The following cases show that in infections of the blood stream the esophagus, like the other organs of the body, can be involved. The first case is one of cavernous sinus thrombosis in a boy (J. B.) aged five, in whom pneumonia and empyema were found at autopsy. In the slides there were numerous areas of infection and necrosis. There was an exudate over the necrotic area, and bacteria crowded

Fig. 18. The photograph shows a fibrous narrowing at the lower end of the esophagus. Above this, for one half of the length of the specimen, the muscles of the esophagus are much thickened.

the exudate and the esophageal tissues beneath. The second case (adult) is one of infectious thrombophlebitis. The slide showed three microscopic periesophageal abscesses and an abscess of the esophageal wall (Fig. 19). The third case is one of mesenteric thrombosis. In this the esophagus was infected, the glands were dilated, and in one place an infected and blocked duct had broken down into an abscess.

Pneumonia. Of thirteen cases of pneumonia, one central, two lobar and ten bronchopneumonia, six showed acute infection, three chronic infection and two periesophageal hemorrhage. The gland ducts were enlarged, probably from infection, in two of the pneumonia cases (Fig. 20).



Empty Abscesses

Fig. 19. Infectious thrombophlebitis. The first photograph shows three periesophageal abscesses. The second photograph shows an abscess in the esophageal wall near the periesophageal abscess.

In the case of central pneumonia the microscopic slides showed three superficial veins obliterated with thrombi (Fig. 21). These were made up mostly of small round lymphocytes.

General Peritonitis. Of three cases of general peritonitis, one showed marked infection of the esophagus.

CHRONIC DISEASE WITH ACUTE INFECTION

There was one case of duodenal ulcer with hemorrhage. The slide from this showed a fibrinous exudate under the epithelium equal in width to the combined thickness of the circular and longtitudinal muscular layers. The exudate showed acute infection.

In one case the patient suffered from starvation, dehydration, and an acute exacerbation of chronic nephritis. The microscopic slides showed acute infection.

Fig. 20. Central pneumonia. The section shows three thrombosed veins.

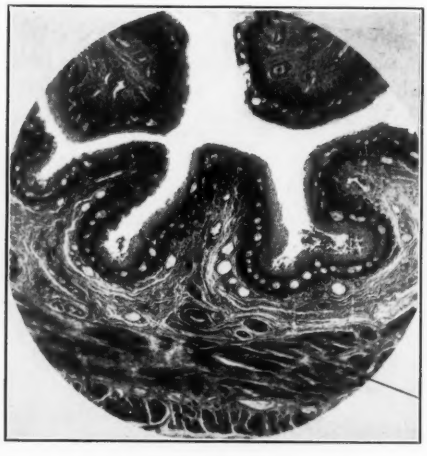


Fig. 21. The section shows enlargement of the superficial blood vessels.

In a case of arteriosclerosis, a specimen taken from the lower end of the esophagus showed acute necrosis of the mucosa of the esophagus with polymorphonuclear leucocytes and bacteria in the necrotic area.

In a case of cholecystitis in which operation was performed, there were multiple abscesses of the liver. The microscopic observations were as follows: There was thickening of the subepithelial connective tissue and a subepithelial infiltration of small round lymphocytes. In places enough polymorphonuclear leucocytes were present to indicate acute infection. There was dilatation of the superficial vessels. A myeloma was found in three of the sections. In this case, therefore, there were both chronic and acute infections.

The slides showed a marked subepithelial infiltration of lymphocytes and greatly dilated vessels. The myelomas were all in the circular layer.

In a case of general arteriosclerosis the slides showed thickening of the subepithelial connective tissue due to a deposit of fibrin. The gland ducts were dilated. The esophagus showed acute infection and ulceration.

Cholecystectomy was performed in a case of pyemia in which there were multiple abscesses of the lungs. Unlike the other cases of infection from the blood stream previously mentioned, the esophagus showed not only acute infection but chronic infection as well. This case is of value because it is another instance of infection of the gall-bladder associated with infection of the esophagus.

FLAKING OF EPITHELIUM

In examining the esophagus after death, it is not uncommon to find areas of discoloration. When the epithelium is unbroken, it is due to enlargement of the superficial veins. For years I considered this condition a postmortem change because the mucosa of the esophagus, like that of the stomach, quickly disintegrates. The esophageal epithelium seems however a little more resistant than that of the stomach. I was curious to see whether this condition might not be due, in certain cases, to infection. Microscopic examination of sections taken from such areas show that it can be.

The following two cases illustrate this condition.

In the first, the diagnosis made at autopsy was coronary thrombosis, intramural thrombus and multiple thrombi. There was a loss of epithelium associated with chronic infection.

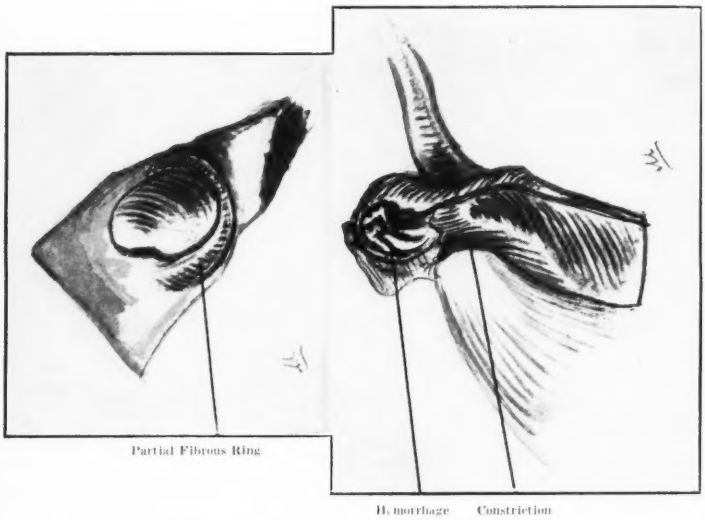


Fig. 22. The patient from whom this esophageal specimen was taken died of gall bladder infection. The opening for the passage of the esophagus through the diaphragm was narrowed and partially surrounded by a fibrous ring. At this point the esophagus was markedly constricted. Just above the diaphragmatic opening there was a large hemorrhage into the esophagus which nearly divided its musculature.

The diagnosis made at autopsy in the second case was renal tuberculosis. Here there was an extensive loss of the esophageal epithelium. The microscopic examination showed an acute infection (polymorphonuclear leucocytes) associated with chronic infection. There was an increase of the subepithelial connective tissue and a marked massing of lymphocytes in the subepithelial stroma.

These cases, if seen clinically, would be classed as acute and chronic esophagitis.

Two more cases remain to be cited in the series selected for the purposes of this paper. The first was a liver case, that of a man who had a prostatectomy and died of bronchopneumonia. Seven inches from the cardia where the esophagus was thin an area of ulceration and infection was present. There was also periesophageal hemorrhage.

The second was a case of purulent ascending pyelonephritis, which showed a subepithelial and periglandular infiltration with small

lymphocytes, and infection under the epithelium, that is, chronic infection.

BLOCKING OF THE GLAND DUCTS BY DOWNWARD GROWTH OF THE SURFACE EPITHELIUM

It has been mentioned earlier that infection of the glands is an important cause of infection of the esophagus itself. The glands are mentioned again to record an observation in connection with them, namely, blocking of the gland ducts by downward growth of the surface epithelium. Two cases of cirrhosis of the liver have been previously cited to show dilatation of the gland ducts from back pressure. Two more cases of cirrhosis of the liver are now mentioned to illustrate blocking of the gland ducts from downward extension of the surface epithelium. This is a new finding in the esophagus which I reported some years ago. This peculiar behavior of the epithelium occurs in the breast in Paget's disease (cirrhous carcinoma). Dr. T. B. Mallory, pathologist at the Massachusetts General Hospital, reported that he had never seen this condition in the esophagus. In the breast it is considered a precancerous change.

Solitary Follicles and the Esophageal Glands. While speaking of the esophageal glands I wish to record another observation, and one that harks back to chronic infection.

In the routine examination of sections of the esophagus, I noticed a cross-section of a gland duct squarely in the middle of a solitary follicle. This was thought to be a chance happening, but further search brought to light many other sections which showed the same finding. This raised the question as to how solitary follicles are formed. It has long been known that they increase in number in infection, especially in chronic infection. It has been supposed that the lymphocytes which form them come from contiguous blood vessels, and it is not uncommon to find collections of follicles about blood vessels which are near the surface of the esophagus. It seems from my findings, however, that they can be formed by collections of lymphocytes about gland ducts.

That these ducts are not always seen in the follicles is due to the fact that in cutting the sections, the duct is missed. A fortunate set of serial sections settled this point. They show a superficial gland which is moderately infiltrated with lymphocytes. Nearby is the dilated duct of the gland with a collection of lymphocytes about it. This duct can be followed to the surface of the epithelium. As it pierces the epithelium, there is a collection of lymphocytes on either

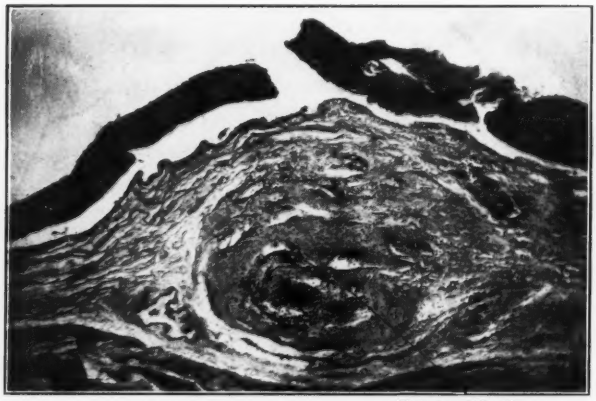


Fig. 23. The illustration shows a large thrombosis of a superficial vessel of the esophagus. Further growth of the thrombus would push off the epithelium of the esophagus and produce a superficial erosion which could readily cause an adhesion or become an infected ulcer.

side of it. The section is cut in the long axis of the duct. If it had been cut horizontally, it would show a section of the duct entirely surrounded by lymphocytes. In other words, if cut horizontally, it would give the picture of a solitary follicle having a section of the duct within it. If, on the other hand, the section had been cut on a slant, the duct would not be visible, and the section of the mass of lymphocytes would appear as the ordinary solitary follicle. This, then, is one method by which a solitary follicle can be formed.

HEMORRHAGE INTO THE ESOPHAGUS AND PERIESOPHAGEAL HEMORRHAGE

The esophagus has an abundant blood supply which is necessary to protect and renew the epithelium. This has a sloppy job to do and is continually subjected to slight trauma and often to excessive trauma. In no other part of the body has epithelium such hard work to do, except at the end of the alimentary canal where again it is often in trouble (Fig. 21).

At autopsy the superficial veins frequently stand out markedly due to postmortem thrombosis. On microscopical section the veins

are not only often found enlarged but areas of hemorrhage about them are common, and these areas are not uncommonly surrounded by infection.

Hemorrhage into the esophagus occurs at birth as part of the birth asphyxia (Figs 10 and 11). The writer feels, however, that in some cases it can be due to sepsis. This belief is strengthened because in one autopsy specimen of an adult esophagus (the patient died of gall bladder infection) there was extensive hemorrhage into the esophagus at the cardia which nearly divided the esophageal musculature. Hemorrhage due to sepsis is an old pathological entity (Fig. 22).

In the adult intramuscular hemorrhage occurs, also periesophageal hemorrhage. Some of the latter is an artifax associated with the removal of the esophagus at autopsy. The writer has found a few examples of single thrombosed superficial veins of extreme size. These were in contact with the epithelium, and a further enlargement would dislodge it and result in a potential ulcer. It has been pointed out that the superficial glands in the upper part of the esophagus are often found bare of epithelium and thus ready for infection. The esophagus like the stomach is an ulcer-forming organ, but to a less extent as shown by clinical symptoms.

The importance of hemorrhage into the esophagus especially in the adult is that where the hemorrhage is extensive enough to cause, in healing, sufficient fibrous tissue, the lumen of the esophagus becomes involved. Hemorrhage into the esophagus might play a part also in cases of rupture of the esophagus.

A nine-inch specimen in a case in which the autopsy diagnosis was adynamic colon showed a slightly dilated esophagus. There were two areas of subepithelial hemorrhage, and near the upper end a hemorrhage which extended through both muscular layers. At the cardia there were subepithelial hemorrhages. I have spoken of these hemorrhages in a previous paper. They are not rare and must be reckoned with in the pathological changes which occur in the esophagus. An instance has just been cited where extensive hemorrhage was associated with sepsis. The probable cause of most cases of subepithelial hemorrhage, however, is back pressure, as in cirrhosis of the liver.

Before giving the closing summary I should like to touch upon two other subjects which, though related to the main subject of the paper, may seem to interrupt its order a little. The first subject is infection of Auerbach's plexus. The English school have held that

Fig. 24. Enlarged drawings of the lower end of an adult esophagus to show segmentation. This is the most extensive case of segmentation which the writer has observed.

fibrosis of the plexus and the consequent loss of ganglion cells was the cause of the condition known so long as cardiospasm. The writer has held that this was a terminal condition; that it was natural in a disorganized esophagus such as is present in long-standing cases of fibrosis of the terminal portion of the esophagus. Nerve tissue has long been known to be resistant to infection. In the hundred specimens which compose the material on which this paper is founded, the writer found only one or two instances in which there was evidence of infection of Auerbach's plexus. This he has found to be true even in cases where the patient had died of the most violent chest infections, for instance, septic pleurisy and pneumonia following perforation of the esophagus by a foreign body or by instrumentation.

Fig. 25. Notice the almost complete disappearance of both muscular layers at the point of the septum, and the increase of fibrous tissue in the longitudinal layer.

A South American observer working partly in London and partly at home reported his findings a few years ago in a series of cases of enlarged esophagus due to obstruction at the terminal portion. His microscopical findings were a loss of ganglion cells in Auerbach's plexus. He attributed this to a food deficiency, very much after the fashion of beriberi. This is an extremely important observation, in fact, the most important one made upon the cause of diseases of the esophagus for many years. It will certainly bear watching especially in tropical countries. Until the etiology of infantile paralysis is fully cleared up one cannot help wondering a little about how this disease produces its occasional effects upon the esophagus.

FIBROUS SEPTA OF THE ESOPHAGEAL WALL

In its early embryological stage the esophagus is a segmented organ. Between the segments the blood vessels gain entrance to the esophagus from the outside. The writer has found in a number of

adult specimens of the esophagus, and always at the lower end, single or multiple fibrous septa dividing the musculature of the esophagus. Their practical importance is that they lie in wait for the tip of a bougie, and invite perforation. They are referred to here because the writer thinks they are another evidence of fibrosis of connective tissue in the esophagus due to infection. (See Figs. 24 and 25.)

SUMMARY

It has been shown that the esophagus, like all other organs of the body, is subject to acute and chronic infection in acute and chronic systemic disease. It can be infected before birth and is also subject to extensive hemorrhage at birth. The most logical explanation for so-called spasm of the pylorus at birth is a light fibrosis due to infection. The three instances cited by the writer where at birth there was a narrowing of both the pylorus and the cardiac end of the esophagus is best explained by fibrosis due to antenatal infection rather than by double spasm.

Infection of the esophagus can originate within or without the esophagus or from the blood stream by an infectious thrombophlebitis. When the esophagus is infected from within, the pathway is probably the esophageal glands. It was shown by other writers many years ago that the superficial glands at the upper end of the esophagus ulcerate easily. Infection can reach these glands readily from the pharynx, a part of the body often subjected to infection, or from the apices of the lungs. Below, the chief source of infection is disease of the liver and the gall-bladder. Autopsy specimens show that even infection of the appendix can be associated with infection of the esophagus. Periesophageal abscess is not uncommon especially at the cardiac end of the esophagus. In the last ten autopsy specimens of the esophagus recently examined by the writer three such abscesses were found.

Dilatation of the superficial veins of the esophagus is very common. Common also is hemorrhage about these veins. The writer has found instances where a single vein was greatly enlarged and thrombosed and was in close contact with the esophageal epithelium. One could easily see that any further enlargement of such a thrombosed vein would tend to push off the epithelium and expose the subepithelial tissue of the esophagus to infection. In the resting state the lower part of the esophagus is closed by the deep vertical folds characteristic of this locality. An ulcer caused by trauma or infection could easily glue these folds together and result in obstruc-

tion. One type of fibrosis of the terminal portion of the esophagus has this relatively slight pathological condition as its cause. Such cases are most dramatic because the gluing is readily broken by instrumentation, and a quick cure of the patient results. Infection followed by fibrosis of connective tissue, to repeat what has been said earlier, is an old pathological entity, and is a logical, a proved, and a common cause of stricture of all grades in any part of the esophagus.

127 FRONT STREET.

XLVI

ACHALASIA OF THE ESOPHAGUS: WITH REPORT OF A CASE RELIEVED BY ESOPHAGOGASTROSTOMY

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The following case of enormous dilatation of the esophagus, which recently came under my observation, is worthy of record because the patient was successfully relieved by esophagogastrostomy.

REPORT OF A CASE

The patient, an asthenic negro, aged 36 years, was first seen in the Out-Patient Department of Cleveland City Hospital in September 1937. He came for treatment of "heart trouble" because he had been suffering from palpitation and precordial pain. The patient had lost 20 pounds during the previous year. There were some abnormalities in the heart sounds, and a diagnosis was made of rheumatic heart disease with mitral stenosis and aortic stenosis and insufficiency.

Fluoroscopic and roentgenographic studies made by Dr. Harry Hauser showed the heart to be of normal size, slightly displaced to the right. The aorta was of normal size and configuration. Administration of barium showed the esophagus to be dilated and filled with retained fluid. The distal end of the esophagus was constricted so that the contrast material failed to pass into the stomach.

The patient was advised to return for additional study of this condition, but did not do so for two years. When he returned, in October 1939, his complaint was that he was regurgitating all his food, and that he had lost 20 pounds in the last month. He had not been feeling well for about five months, but had continued to work until a month before admission. He said that he had been vomiting almost all his food, immediately after eating. He said that he could feel the food "getting stuck" at the lower end of the sternum. He could take fluids better than solids, and these aided in keeping some solid material down. There was no history of hematemesis or melena. The patient said that he had had six other episodes similar in nature, the first in 1923, but that this last attack had been the most severe. His weight was 98 pounds; he had lost 37 pounds in three years. The temperature was 38.4 degrees C.; the pulse rate was 84; the respiratory rate, 22; and the blood pressure, 108 systolic, 62 diastolic. Except for the extreme emaciation, there was nothing remarkable in the physical examination, aside from the chest. The lung sounds at the apices were distant. Precordial activity was increased visibly. The apex impulse was in the sixth interspace in the nipple line. There was a systolic murmur over the apex and a clapping second sound. There was also some aortic murmur and a questionable split first sound at the apex. The fingers and toes were clubbed.

The erythrocyte count and hemoglobin were normal, but the leucocyte count was high, 35,000. The differential count showed 77% polymorphonuclears, 20% lymphocytes, 3% monocytes. The serologic test was negative.

Roentgenographic examination of the chest showed a widening of the mediastinum to the right, extending from the level of the second thoracic vertebra to the diaphragm. The patient swallowed two glasses of barium in rapid succession without filling more than the lower third of the esophagus. The contrast material passed through the cardio-esophageal orifice very slowly at first and then somewhat more rapidly later. About five minutes were required to allow enough barium to enter the stomach for satisfactory examination of this organ. No abnormalities were found in the stomach. The duodenal bulb filled rapidly, and appeared normal.

The condition was regarded as cardiospasm, in view of the long history, and the diagnosis of rheumatic heart disease, made at the previous admission, was also reaffirmed.

Esophagoscopic examination showed that the esophagus was tremendously dilated and contained about 2 liters of malodorous material. The esophageal wall was markedly thickened and granular. The diaphragmatic opening could not be found.

Following this, esophageal lavage was ordered, and was carried out every day for a week or ten days. The patient then was transferred to surgery, and after another ten days, operation was performed by Dr. Freedlander. First, the phrenic nerve was crushed, and then esophagogastrostomy was performed. The lower end of the esophagus was brought down through the hiatus of the diaphragm for a distance of about 3 cm. Then the serosal surfaces of the esophagus and the cardiac portion of the stomach were approximated by interrupted silk sutures. The line of union extended in a diagonal direction from the upper left portion of the esophagus to the lesser curvature of the stomach. A U-shaped incision was made from the cardia of the stomach, extending down through the mucosa. The base of the U curved around the right end of the previous line of sutures, and the upper arm was carried out through the cardio-esophageal junction, ending on the lower portion of the esophagus. The tongue of tissue composed of a portion of the esophagus and stomach at the cardia was then inverted so that the serosal surfaces were in contact, and continuous sutures were applied. At the outer angles of the U, the corner was carefully turned, and the outer serosal edges brought together by means of a continuous chromic suture. Continuous suction was applied while the anastomosis was made. Drains were inserted in the line of anastomosis and brought out through the wound.

The patient's course was somewhat stormy for three weeks, and there was some infection in the upper portion of the wound, but when he began to improve and to have a high vitamin, high calorie diet, he gained 10 pounds in the course of a week. Roentgenograms made six weeks after the operation showed approximately the same degree of dilatation, but there was improvement in the rate of flow of the barium through the cardiac orifice, which was still constricted. Fluoroscopic studies a month later (two and one-half months after operation) showed that there was still some retardation of the flow of barium at the distal end of the esophagus. This appeared to be due to diminished contractility of the esophagus and a failure of normal propulsion. There was some medial displacement of the cardia and a shadow which suggested some diaphragmatic and perigastric peritoneal thickening.

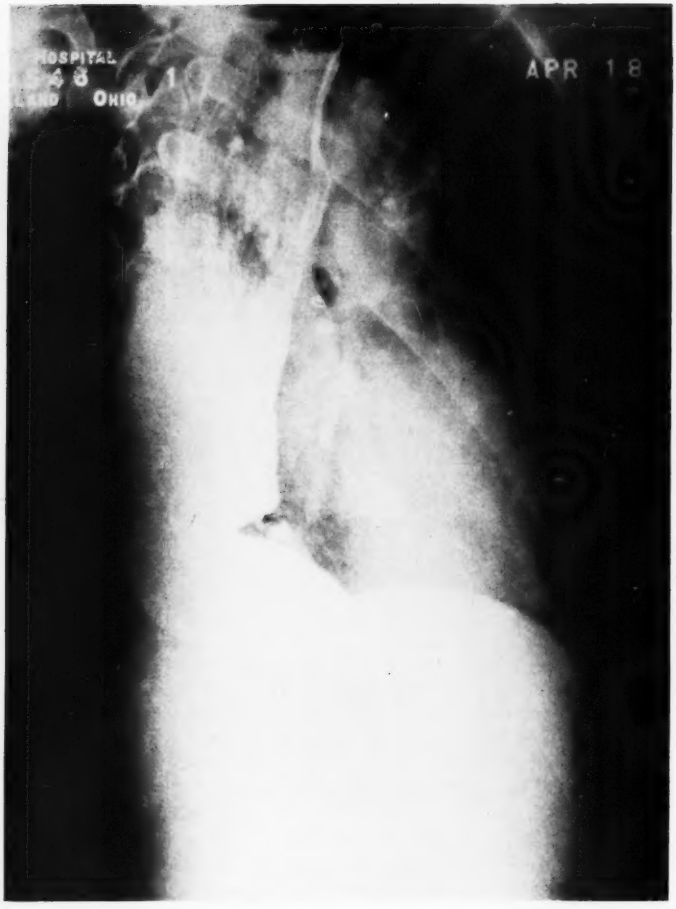

Fig. 1. Roentgenogram of the esophagus filled with barium, showing marked dilatation and tortuosity in the distal third and tapering, smooth constriction of the distal end.

The patient was discharged a few days later. He was in good physical condition, although the drainage tubes were still in place. At a subsequent examination in the out-patient department, two weeks later, the drainage tube was shortened and reinserted. The patient had no symptoms and was able to eat well.

COMMENT

The results in this case prompted me to investigate the literature of the last few years on achalasia to see whether any similar case had been recorded. I did not find any report in which the details of the case history were strikingly similar to this case, but a number of interesting facts were elicited in this study which it seems worth while to review.

Etiology.—The etiology of cardiospasm is still obscure. Since authorities differ considerably also as to the pathology, it is extremely difficult to account for the cause of this condition. In clinical experience and in studying the literature, however, one cannot help but be impressed that the most prominent and consistent factor in these cases is nervousness and shock. So many of the patients are nervous and high strung, and a review of the recent literature discloses that cardiospasm developed following accidents or operations in too large a proportion of cases to be merely coincidental. Vinson[1] reported four cases of cardiospasm or esophageal lesions which occurred following severe accidents in which there was no injury to the thorax. Stevenson's[2] case was in a man who had had mild symptoms for a good many years, but these were markedly aggravated by an accident which occurred when his horses ran away while he was plowing. Cases which followed operation have been reported by Behrend[3] and by Benedict and Daland.[4] These cases suggest that the shock occasioned by accident or surgical operation may have initiated the disturbance in the esophagus. This implies some disturbance in the sympathetic, adrenal, parasympathetic mechanism. In the cases in which there is no history of sudden shock, this mechanism may have been upset by some other strain such as overwork, worry, or some endocrine imbalance.

It is well recognized that control of nervousness and rest are of importance in the treatment of cardiospasm. Starling and Camps[5] have reported a case in which complete symptomatic relief was obtained in a patient with severe pressure symptoms by rest in bed and the administration of belladonna. In describing a case in an eight year old girl, Tallerman[6] observed that vomiting was less marked when she was resting, and during one stay in the hospital when she was kept in bed, she scarcely vomited at all; yet as a rule, no more

than two or three days passed without severe symptoms. In a series of 17 mild cases of cardiospasm, all of them in highly nervous individuals, Schmidt[7] reported good results obtained by the administration of drugs such as stramonium and papaverine. He also commented that in some cases the attacks were precipitated by emotional or nervous strain.

That there may be an endocrine factor in the causation of cardiospasm is suggested by the case reported by Levin.[8] The patient was an aged man, 80 years old, who had periodic violent tetanic muscular contractions during which he could not swallow even water. Blood studies revealed a marked calcium deficiency. With the administration of parathyroid hormone and calcium intravenously, the seizures ceased immediately, and water and soft food could be taken without difficulty. Contractions continued to occur, however, until there was a full stabilization of the blood calcium. The author raised the question as to whether this patient suffered from calcium deficiency throughout the period of 32 years that he had had dysphagia, and whether the calcium disturbance was partially or entirely responsible for the cardiospasm. He also asked whether a calcium disturbance even in a mild degree may be responsible for cardiospasm, and if so, whether fatigue, exhaustion, worry and nervousness produce a a calcium deficiency, or vice versa.

Another interesting case in which allergy apparently was the cause of cardiospasm has been reported by Withers.[9] The patient had severe symptoms typical of cardiospasm, and was allergic to chocolate, peanuts, and string beans. The symptoms could be produced at will by ingestion of these foods, and the attacks were completely controlled by the administration of adrenalin.

Pathology.—It is not easy to gain a clear idea of cardiospasm from the pathologists, for they do not agree about the findings in the esophagus. Mosher[10] has come to the conclusion that cardiospasm is the result of an inflammatory process which produces fibrosis in the terminal portion of the esophagus. He has seen 30 consecutive cases of cardiospasm which show a crural crease in the esophagus. He believed this to be due to a fibrosis of the connective tissue in the crural ring associated with a fibrosis of some of the muscle bundles of the esophagus and of the crura. This he attributed to infection from neighboring organs. In three cases, at necropsy he found extensive fibrosis of the terminal or strictured portion of the esophagus, with no marked fibrosis above this point. He suggested that fibrosis of the terminal portion of the esophagus would be a better term than cardiospasm.

On the other hand, Lendrum,[11] in a study of 13 cases postmortem, came to quite different conclusions. He stated: "In all cases the dilated portion of the esophagus or sac was separated from the stomach by an undilated segment of esophagus which I have labeled the neck. The neck varied in length between 1.5 and 4.5 cm. In no case was there organic narrowing of the neck or scarring in or around it. Accordingly one may lay the ghost of Mosher's 'fibrosis of the terminal esophagus.'" The one consistent finding in Lendrum's series of cases was a striking loss or complete absence of ganglion cells from the myenteric plexus. Sometimes there were new supporting cells filling the capsule or an extinct ganglion cell. Sometimes there was inflammation in and about the ganglia. Thus he stated that his findings were compatible with the theory of Hurst that cardiospasm or achalasia of the cardia is primarily a disease of the myenteric plexus. This theory is that the destruction of the myenteric plexus allows the sympathetic fibers to the terminal portion of the esophagus to act unopposed. As a result the terminal portion of the esophagus does not relax normally with the swallowing reflex and dysphagia results.

Lendrum stated the belief that in early and moderate cardiospasm the esophagus responds to the obstruction by slight dilatation and increased thickening of the circular muscular wall, which he called compensated cardiospasm. Long-continued and severe pressure overcomes the resistance of the esophagus to dilatation so that it becomes dilated and stretched lengthwise, and bends and kinks may occur.

Another recent pathologic study in cardiospasm is reported by MacCready[12] in two cases. He said that chronic inflammatory changes were the most prominent finding, and felt that his findings confirmed Mosher's opinion. He added, however, that there was no intra-abdominal infection, and that it was not possible to rule out a neurogenic origin of the condition. He said there was no evidence of hypertrophy of the muscle fibers at the cardia, and the infection was much less evident in this portion.

These somewhat contradictory conclusions are at least partially resolved by Turner's[13] statement concerning the pathogenesis of cardiospasm. "Although cardiospasm or achalasia is not usually associated with gross structural change in the wall of the esophagus, this condition may be a direct cause of such a change, in view of the cases. In the first instance, it is possibly simply a neuromuscular defect, but as a consequence dilatation of the esophagus occurs and

there is some resultant stasis. In some cases this stasis in turn leads to inflammatory changes in the mucous membrane which may extend to the deeper parts, and once that has occurred, the development of fibrous tissue and subsequent organic stenosis may readily follow."

Diagnosis.—Although, as a rule, the diagnosis of cardiospasm is not difficult, there are some instances when considerable confusion may arise. The history and roentgenograms and fluoroscopy usually indicate the proper diagnosis, but these may be misleading at times. In a few cases, it is difficult to differentiate between neoplasm and tremendous esophageal dilatation, especially when the secondary symptoms produced by the dilated esophagus are particularly prominent.

Cardiospasm can usually be differentiated from carcinoma of the esophagus by the history. Freeman, in discussing a presentation by Vinson,[14] summarized the differences in the two conditions as follows: "The onset of dysphagia is slow and gradual in carcinoma with the first symptom, difficulty in swallowing solid food. The patients with carcinoma are usually men more than 40 years of age. In cardiospasm, there are just as many women as men, and the patients are of any age. The difficulty in swallowing is sudden and as likely to result from liquid as from solid food."

Wright,[15] in discussing the diagnosis of lesion of the esophagus, also stressed the importance of the history. He noted that difficulty in swallowing solid foods was more likely to accompany malignant lesions, while greater difficulty in swallowing liquids usually is a sign of cardiospasm. This author also called attention to the pain simulating angina pectoris in chronic cardiospasm. Substernal or precordial pain radiating through to the back may precede dysphagia in many cases of ulcer of the esophagus. There also is precordial pain radiating to the left upper abdomen in diverticulum of the middle third of the esophagus.

According to Clerf,[16] cancer of the cardiac end of the stomach which involves the esophagus secondarily may simulate cardiospasm, and may be a source of confusion. The roentgenologist commonly designates the condition as cardiospasm. At esophagoscopy, however, the narrowing at the hiatal level does not permit the passage of the esophagoscope, and a diagnosis of infiltrative stenosis must be arrived at. If the growth intrudes through the esophageal wall, a biopsy can be made. If this is not possible, the diagnosis must be based on the presence of infiltration and fixation. Clerf has observed several

cases of this type in which surgical investigation revealed carcinoma of the cardiac end of the stomach.

Other conditions besides carcinoma which must be differentiated from cardiospasm are cicatricial stricture, diverticula, hernia of the stomach through the esophageal hiatus in the diaphragm, foreign bodies in the esophagus, and congenital stricture. Clerf[17] recently reported a case in which a tuberculous periesophageal abscess produced stenosis and extreme dysphagia, which was relieved when the abscess was drained. A history of having swallowed some corrosive substance usually points to cicatrical or benign stricture, although this condition may be present in the absence of such a history. Vinson[18] says that the cause cannot be determined in 20% of the cases. Apropos of corrosives, it is interesting that Sagel[19] has reported a case in which the offending substance was a pint of moonshine, which presumably contained a large quantity of lye. Diverticula are usually easily recognized by roentgenographic examination. Foreign bodies may be determined by the roentgenogram, but in some instances, esophagoscopy is essential in making the differential diagnosis.

Cases in which cardiospasm was mistaken for mediastinal tumors by the roentgenologist have been reported by Starling and Camps,[5] Behrend[3] and Chandler.[20] Starling and Camps' patient, before they saw her, was subjected to a course of X-ray treatments on the basis of the mistaken diagnosis. When the true nature of the condition was recognized, the patient was cured by retrograde dilatation of the cardia through an opening in the stomach.

The pressure of the esophageal dilatation may produce secondary pulmonary symptoms which lead to faulty diagnoses, masking the true condition. In the case reported by Schmidt[21] in a six year old girl, the diagnosis was aspirational pneumonitis, which could not be controlled until dilatation of the esophagus had been carried out. DeVeer[22] reported a case in which, for ten years, the diagnosis had been *pleuritis mediastinalis*, because the condition was associated with periodic attacks of fever and pulmonary symptoms.

In such cases as these, and in other cases in which the diagnosis is not clear or the extent of the difficulty is obscure, esophagoscopy is extremely useful in making an exact diagnosis. In the case I have reported, the esophagoscopic examination revealed the extent of the dilatation and the quantity and nature of the retained fluid. Schmidt[23] has cited the value of esophagoscopy in diagnosis with four interesting cases. The first was an atypical case of carcinoma of the esophagus at an advanced stage. There was little evidence of steno-

sis at the site of the lesion and no subjective sensation of solid or liquid food sticking in the esophagus. The second was a case in which there was complete esophageal obstruction in a case of cardiospasm. In the third, a stricture developed at the esophagogastric junction of a congenital short esophagus. In the fourth case, a wild plum pit had become impinged in a benign stricture of the esophagus. The stricture had developed many years previously after a severe illness with typhoid fever. The foreign body was suspected, but no accurate history of it could be obtained until after esophagoscopy.

Treatment.—The treatment of achalasia or cardiospasm is medical, mechanical, or surgical. The choice of treatment varies with the preference of the individual physician or surgeon, as well as with the severity of the condition.

Numerous authors have stressed the importance of rest and sedative drugs in controlling cardiospasm. Starling and Camps[5] reported a case of severe cardiospasm which was relieved completely by rest in bed and the administration of belladonna. Schmidt[7] has described good results in 17 cases of functional spasm of the esophagus in which there was not extreme dilatation, by the use of stramonium and papaverine. In Withers'[9] case, ascribed to allergy, the condition was controlled by the use of adrenalin. The patient described by Levin[8] was relieved completely by the administration of parathyroid hormone and calcium. These case reports furnish material for thought and suggest that various forms of medical treatment may be of benefit in properly selected cases of achalasia.

Canfield[24] has stressed the importance of irrigations to correct inflammation of the esophagus before dilatation is undertaken. In the case I have reported, this procedure was of distinct importance in preparing the patient for surgical treatment.

Dilatation of the esophagus, performed by various instruments and procedures, is recommended by a majority of authors as the treatment of choice for cardiospasm. Although no attempt will be made here to discuss the various types of dilators, one of the most recent instruments devised for esophageal dilatation by Browne and McHardy[25] might be mentioned. This combines the principles of Hurst, Plummer and Smithies, and consists of a mercury tube at whose distal end is incorporated a rubber-covered silk bag, to which a small catheter runs through the mercury-filled tube. Distention is effected under the control of a sphygmomanometer.

Tallerman,[6] Behrend,[3] Chandler,[20] Schmidt,[21] Mosher,[10] Benedict and Daland,[1] and Vinson[14] have described cases of extreme dilatation

of the esophagus due to cardiospasm which were relieved by mechanical dilatation. In cases of benign stricture of the esophagus, Vinson[18] has stated that gastrostomy seldom is necessary, and he cited the case of one patient in whom dilatation was accomplished successfully despite the fact that he had had symptomatic obstruction of the esophagus for eight years.

In cases in which medical and mechanical treatment fail to relieve the patient, some form of surgical interference is advisable. According to Knight,[26] these surgical procedures fall into three classifications: (1) plastic operations on the esophagus, (2) plastic operations on the cardia, and (3) short-circuiting operations. To these procedures, he added a fourth, sympathectomy, which is useful only in true achalasia and cardiospasm, but not in hypertrophic stenosis of the cardia. Heyrovsky,[27] who has contributed a lengthy review of the subject of cardiospasm to the German literature, comes to the conclusion that in mild cases dilatation and the cardiomyotomy described by Heller are to be recommended, and that in severe cases, subphrenic esophagostomy should be employed.

The operative procedures that have been described by different authors in the recent literature vary considerably. Schaffner[28] has reported a successful case in which esophagoplasty was performed. Starling and Camps[5] and also Stevenson[2] reported successful results by opening the stomach and dilating the cardia with the fingers.

In the case reported in this paper, a short-circuiting procedure, esophagogastrostomy, was employed. In a recent report, Heatley[29] reported successful results in two cases of cardiospasm with a similar operation. One of his patients was a woman, aged 79 years, who had had symptoms of dysphagia for seven or eight years; these had become progressively worse during the last two years. The second patient was a woman, aged 25 years, who had suffered from dysphagia for seven months. Numerous dilatations failed to benefit her, and she had lost about 25 pounds and was very weak and nervous at the time the operation was performed. Heatley has furnished a good review of the use of esophagogastrostomy for the relief of intractable cardiospasm, beginning with the work of Heyrovsky in 1910 and including the cases reported by Bull, Turner, Churchill and Womack. From my own experience, I agree with Heatley's conclusions and his recommendation of this type of surgical procedure for the relief of severe cardiospasm which cannot be controlled by more conservative measures.

Preliminary reports on the results of sympathectomy have been made by Knight and Adamson,[30] who felt that their early attempts

were sufficiently encouraging to warrant further trial. Knight[25] concluded that sympathectomy was of value only in cases of true cardiospasm or achalasia, that is, those with a functional basis or background. Sympathectomy had no effect in a case of hypertrophic stenosis of the cardia. For these reasons, he urged more careful study and diagnosis of cases of this type, in order that they may be subjected to rational therapeutic procedures.

From my own experience and from the reports in the literature, it would appear that, even in the cases in which dilatation of the esophagus is most extreme and obstruction practically complete, there is hope for relief of the patient by surgery. The good results in the case reported here and in the others cited should encourage surgeons to resort to similar measures in extremely severe cases.

It goes without saying that if less radical methods afford relief, surgical intervention should not be considered. Whenever mechanical dilatation is possible, and it is possible in some very severe cases, this is the treatment of choice. In view of the results reported in some instances following rest and medical treatment, it behooves all of us who are working in this field to investigate these possibilities further, and perhaps eventually to work out by these means some more satisfactory classification of cases of cardiospasm or achalasia.

SUMMARY

A case of extreme dilatation of the esophagus in which the condition was satisfactorily relieved by esophagogastrostomy is reported. The patient was a negro, aged 38 years, who had had symptoms of dysphagia for 16 years. During that period, he had had five or six episodes in which the symptoms were extremely severe, with remissions between. During the last two years before admission, he had lost 37 pounds, the lower end of the esophagus was enormously dilated, and practically no food passed into his stomach. After the operation, the defect was not entirely corrected, according to roentgenographic examinations, but the patient was able to eat well, and gained weight and strength rapidly.

This case, and several others reviewed in the literature of the last few years, show that extreme instances of achalasia may be relieved by surgery, when mechanical dilatation is impossible.

Many of the cases reported recently point strongly to the importance of shock and nervous tension in the causation of cardiospasm. Allergy and endocrine factors also have been implicated in

some of the cases reported. Several patients have been treated successfully by medical means, such as rest and sedatives.

These reports should stimulate all those working in the field to study each individual case with all these facts in mind, with the hope that from such investigation, some more rational classification of cases may be brought forth to influence the results of therapy.

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XLVII

THE EARLY DIAGNOSIS OF CARCINOMA OF THE ESOPHAGUS*

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Statistics for the U. S. A. show that 1.8% of all cancer deaths are due to carcinoma of the esophagus. It has been estimated that if the condition were diagnosed correctly the percentage would be considerably higher. According to Dormanns'[1] out of 23,139 deaths 8% were due to carcinoma of the esophagus. It was fourth in frequency of malignant tumors in men over 20, being surpassed only by carcinoma of the stomach, lung and rectum. According to vital statistics for Chicago in 1938 which showed 4871 deaths from tumors, the number of deaths from cancer of the esophagus, if estimated at 4% would be nearly 200. Eighty per cent of cases have been found in males (Cancer Memorial Hospital, New York), and all but about 10% occur in the middle or lower esophagus, with squamous cell carcinoma the histological type in 90%.

The mortality rate in cancer of the esophagus has been until very recently practically 100%. Treatment has either been palliative, consisting of suitable diet, dilatations, intubations, or gastrostomy, or it has been confined to radiation. In the latter type of treatment both intra-esophageal radiation with radium or radon, and deep X-ray therapy have been used extensively, but results have been extremely poor. Palliation has been produced in a few cases with prolongation of life to as long as two years, and in rase cases longer, but results as shown by statistics have been universally poor.

The failure of radiation therapy is to a large extent to be explained by the pathological characteristics of the tumor in that it produces symptoms late, while metastasizing early, and is usually of the type classed as radioresistant (Watson[2]). Also the deep location in the body with the close relation to vital structures makes adequate radiation impossible without the danger of necrosis and perforation.

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The great progress in the development of intrathoracic surgery in the past two decades has made surgery of the thoracic esophagus a possibility and has revived interest in this form of treatment. Surgical removals of cancer of the esophagus are steadily increasing in number, and operative mortality is steadily decreasing.

Since 1938 the surgical removal of cancer of the esophagus has been carried out successfully in about 50% of operable cases, and over 58% of the successful cases were still living, some as long as four years (Adams). Thoracic surgeons are of the opinion that there is good prospect for further reduction in operative mortality. With the perfection of surgical technique the possibility of cure comes to depend upon early diagnosis, before metastases have formed and before adjacent structures have been invaded.

With the object of obtaining some information as to the possibility of making an earlier diagnosis in this disease a review of the cases of cancer of the esophagus seen in the University of Chicago Clinics in recent years has been made. An average of 10 to 12 cases per year are treated at the clinics and of these, 60 have been reviewed.

CANCER OF ESOPHAGUS—60 CASES

Sex—80% Males.	Age—50% under 60 Years.
Location	
Upper third	19%
Middle	44%
Lower	28%
Stomach and lower third of esophagus	9%
Type	
Squamous cell	80%
Adenocarcinoma	20%
Initial Complaints	
Difficulty in swallowing	53%
Obstruction	32%
Pain in swallowing	28%
Regurgitation	17%
Hoarseness	8%
Other complaints such as anorexia, cough, hematemesis, vague discomfort, weight loss, each about	2%
Time Between Initial Symptoms and Biopsy	
Under two months	17%
Two to four months	32%
Over four months	51%
Duration of Life from Appearance of Initial Symptoms	
Six and one-half years	1 case
Over two years	3 cases
Under one year	82%

Six cases treated by resection are included. Three are living, one for over two years. Thirty-one were treated by radiation, with palliation for over two years in two.

Metastases or local extension of the tumor were diagnosed at the initial examination in over 20%, and were present in a considerably greater number, as revealed by surgical exploration. Spread by lymphatics is most common, the lymph flow being toward the mediastinal and supraclavicular glands from the upper two thirds and toward the cardiac glands along the lesser curvature from the lower third. Distant metastases have been reported absent in 40% (Dormanns[1]) and 35% (Huenermann and Eberhardt[4]) at necropsy. Direct involvement of surrounding structures have been found in about 50%. Perforation of the respiratory tract, erosion of the wall of the aorta, and involvement of recurrent laryngeal nerves are common.

Because the onset is insidious and symptoms are absent usually until some discomfort or interference with swallowing is produced, the outlook for an early diagnosis is limited. However, the review of histories just presented indicates that in over half, the disease might have been recognized at least four months earlier. Recent surgical experience seems to indicate that a fairly high percentage would present the possibility for surgical cure, at the time when symptoms first became evident.

The hope for early diagnosis lies in making a thorough examination in every case with even mild discomfort, pain or difficulty in the act of swallowing. This includes an X-ray examination by fluoroscopy and spot films to show abnormalities during the passage of the barium, and an early esophagoscopic examination.

Esophagoscopy should not be reserved for cases with an obvious obstruction, but must be used early as a diagnostic aid in the presence of mild complaints. In performing the examination there are three essential considerations: The adequacy and thoroughness of the examination, the safety of the patient, and the amount of discomfort caused.

The first of these depends upon the skill and experience of the examiner. The procedure can be safely carried out under local anesthesia in most cases, but under some circumstances such as a short thick neck, extreme apprehension, or failure to cooperate, general anesthesia provides greater safety and may even be necessary in order to make the procedure possible. When local anesthesia is used the

comfort of the patient should receive careful consideration. The fact that the procedure has frequently caused great apprehension and distress to the patient has frequently resulted in a considerable degree of reluctance on the part of the profession to recommend esophagoscopy, which must be overcome if more early diagnoses are to be made. Apprehension and distress may usually be avoided if the preparation of the patient is adequate.

Several methods of premedication have been used in our bronchoscopic clinic: Nembutal, seconal, and avertin have been tried, and while each proved useful in some cases the results were not uniformly satisfactory.

The method preferred consists in the use of morphine and scopolamine, along with a moderate dose of a barbiturate such as nembutal or seconal. The dosage is varied with the individual patient, and should be regulated so that cooperation is still obtainable for anesthetization.

The local anesthetic which we have found most efficacious is cocaine in 10% solution applied either by a spray or on pledgets held in the pyriform fossae, or a combination of the two. Two sprayings followed by a pledget held in each pyriform fossa is usually adequate. Pontocaine has also been used in 2% solution, but in our experience is less efficient when used in amounts which are within the limits of safety. A certain amount of training and practice are necessary for safety in passing instruments into the esophagus. Lack of practice can be compensated for by the use of general anesthesia. The diagnosis of esophageal conditions by direct examination requires greater experience than the actual technique. In certain types of tumors the diagnosis could scarcely be mistaken, but in others, as well as in various inflammatory or post-inflammatory conditions, the examination requires great care and skill.

In the case of tumors or lesions which may be suggestive of neoplasm a biopsy is essential. Certain dangers from taking a biopsy must be guarded against, particularly perforation of the wall or in conditions such an aneurysm or varices, severe uncontrollable hemorrhage.

The squamous cell tumors (80% in this series) present two characteristic types, which on the basis of gross arrangement alone may be described as medullary and scirrhus.

The medullary type grows into the lumen of the esophagus, rapidly producing a mass of tumor easily recognized on esophagoscopy. (See Fig. 1.)

Fig. 1. Fig. 2.

Fig. 1. Section through a squamous cell carcinoma of the espohagus of the type which grows freely into the lumen. The whole thickness of the wall is involved. (Removed by Adams and Phemister.[5])

Fig. 2. Section through scirrhus type of squamous cell carcinoma. Extension occurs both in the circumferential and longitudinal directions, producing early stenosis, with marked hypertrophy of the esophageal wall above the tumor. Biopsy frequently fails to show tumor unless care is taken to get below the hypertrophied wall above. (Removed by W. E. Adams.[3])

The scirrhus type (Fig. 2) extends in the wall of the esophagus both longitudinally and around the lumen. The walls of the esophagus above the tumor become greatly hypertrophied, with a thickened epithelial layer. The characteristic appearance on esophagoscopy is that of firm, pale, edematous-looking folds which obstruct the passage of the instrument as well as the view before the tumor is reached. Biopsy is frequently negative unless an effort is made to get down sufficiently to reach the tumor. Fortunately the hyper-

trophy of the wall provides some protection against perforation in the attempt to reach tumor tissue.

The adenocarcinoma is usually found in the lower third, arising from the gastric type of mucosa in the esophagus, and is easily recognized and biopsied. In the case of carcinoma of the stomach invading the lower end of the esophagus, diagnosis may be difficult by means of the esophagoscope.

SUMMARY

1. Statistics show that carcinoma of the esophagus accounts for 2 to 8% of tumor deaths. It has been universally fatal.

2. Recent advances in thoracic surgery have resulted in an increasing number of successful resections of the esophagus for carcinoma. Further improvement is to be anticipated.

3. Surgical removal can be successful only in relatively early cases.

4. The earliest symptoms of progressive discomfort or difficulty on swallowing are an indication for thorough examination by X-ray and esophagoscopy.

5. Esophagoscopic examination is simple, safe, and without great discomfort when properly managed. It should be done as an early diagnostic procedure.

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XLVIII

COMPLICATIONS OF ESOPHAGEAL PERFORATIONS*

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Perforations of the esophagus are recognized for their multiple, extensive, rapidly progressive complications. The severity of the involvement following a perforation of this viscus is due in part to the number of vital structures immediately adjacent to the esophagus, which may, consequently, be perforated at the same moment the esophagus is perforated. The most common complications of perforations of the esophagus are infection, namely, esophagitis, mediastinitis and septicemia; hemorrhage; perforations of the pleura with resultant pneumothorax and pyopneumothorax; perforations of the trachea and bronchi; emphysema; and pericardial perforations leading to pneumopericardium and pericarditis. Any of these conditions may, of course, prove fatal. The high morbidity and mortality rate is due in part to the fact that the esophagus is surrounded by a potential space of loose connective tissue which provides an extensive area for the rapid, unobstructed advance of infection. The anaerobic conditions in this space, the relative surgical inaccessibility of the major portion of the esophagus and the difficulty in accurately establishing the diagnosis of perforation are further reasons for the severity of the complications.

ANATOMICAL CONSIDERATIONS

In the neck the esophagus lies immediately posterior to the trachea, and on either side of it may be found the carotid sheaths with the vessels they contain. The upper esophagus is bounded posteriorly by the vertebral column and its muscles, but leaves this position at the level of the bifurcation of the trachea, where it at first lies in front of the azygos vein and then, more inferiorly, in front of the aorta. The trachea still lies anterior to the esophagus in its upper thoracic portion, while farther down, below the level of the fifth

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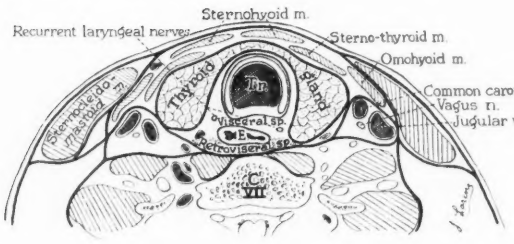


Fig. 1. The relationship of the esophagus to surrounding structures in the neck at the level of the seventh cervical vertebra.

thoracic vertebra and the tracheal bifurcation, the pericardium and left auricle of the heart lie anterior to it. The upper thoracic esophagus is bounded laterally by the pleura. The aorta lies to the immediate left of the esophagus in the mid-thoracic portion, rotating to a more posterior position in the distal esophagus. On the right side, the azygos vein lies in close proximity to the esophagus, migrating posteriorly in the more dependent portions of the posterior mediastinum. In the thorax the esophagus passes through the superior mediastinum and posterior part of the inferior mediastinum.

The anatomical relations of the fascial planes and the spaces surrounding and lying posterior to the esophagus are of utmost significance in relation to esophageal perforations. These fascial spaces which are so anatomically complicated were thoroughly studied and accurately described more than 50 years ago, as Pearse[1] points out, by Henke,[2] König and Riedel,[3] Soltmann,[4] Poulsen,[5] and Schmitt.[6] More recently, Mosher,[7] Furstenberg,[8] Iglauer,[9, 10] Weintraub[11] and many others have made significant contributions to the anatomical descriptions and classifications of these spaces, subdividing them into smaller compartments.

In approaching this subject from the clinical aspect of the management of perforations of the esophagus, certain major compartments must be recognized and variations of divisions of these considered to some extent, at least, as individual variations. Thus, the space surrounding the esophagus and pharynx may be considered as one continuous potential fascial space extending from the level of the nasopharynx to the diaphragm. This potential space is divided arbitrarily into three major anatomical divisions, the uppermost being designated as the visceral space extending from the base of the skull

to the superior aperture of the thorax. It is bounded posteriorly by the prevertebral fascia and anteriorly by the pretracheal fascia. This space is frequently further subdivided, clinically, into a retropharyngeal and retro-esophageal portion. The second major division is the space surrounding the esophagus in its course through the superior mediastinum. This is designated as the superior mediastinal space, and is limited inferiorly at its junction with the posterior part of the inferior mediastinum by the great vessels, bifurcation of the trachea and lung roots. According to Lambert and Berry,[12] it is at this partial barrier to the inferior extension of infections that, as the fascia becomes firmly adherent to the trachea, bronchi, aorta and pericardium, the inflammatory process may extend between the leaves of the pulmonary ligaments and along the bronchi. Consequently, as such inflammatory processes in the mediastinum produce a thickening of these potential spaces there is an increase in hilar markings or "root shadows" which are significant in the roentgen interpretation of pathology in this area.

The posterior part of the inferior mediastinum, lying below the area just mentioned and posterior to the esophagus from the fifth dorsal vertebra downward, is the third portion of the retro-esophageal space. Perforations into this space may lead to infections traveling inferiorly through the diaphragm, retroperitoneally along the psoas muscles.

ETIOLOGY

Neoplastic disease is the most common etiological factor of esophageal perforations. The extension of a carcinoma from the esophagus into the trachea or into the pleural cavities may take place by direct extension of the growth or as the result of the necrosis it produces in adjacent structures. In either case, perforation due to a neoplasm is usually a gradual, chronic type of perforation, occurring in the far-advanced stage of the disease, and little but palliative measures may be used therapeutically. Such measures as gastrostomy or the careful guidance of a feeding tube into the stomach may be indicated to alleviate the symptoms and severity of aspiration pneumonia.

Trauma is second in importance as an etiological factor of esophageal perforation. External trauma is rather rare because of the protected position which the esophagus occupies. The esophagus is seldom cut during the usual procedure of "cut-throat" but may be injured by stab wounds or gunshot wounds. A series of such cases was recently reviewed by Jensen.[13] Accidental surgical per-

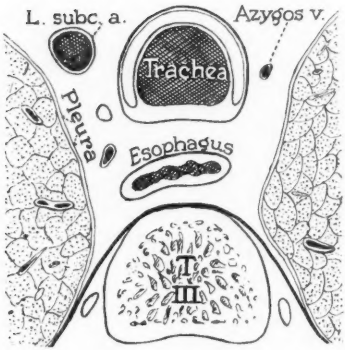


Fig. 2. The position of the esophagus at the level of the third thoracic vertebra.

forations from without are likewise rare but may occur during the operation of tracheotomy, when there is a marked anteroposterior tracheal collapse during an obstructed inspiration. Should the incision through the anterior tracheal wall be made on deep inspiration, incision of the posterior wall or anterior esophageal wall is not impossible. An ill-fitting trachea tube may also produce an esophageal perforation by impinging on the posterior tracheal wall and producing a decubitus ulcer.

Perforations resulting from trauma within the esophagus, on the other hand, are more common. Foreign bodies, instrumentation during the removal of foreign bodies, bouginage, or the simple introduction of the esophagoscope itself may result in a perforation. According to Chevalier Jackson,[14] the introduction of the esophagoscope is "a surgical procedure which has difficulties and dangers which may be classified as educational, anatomic, physiologic and pathologic". Jackson further states that "the esophagoscopic tube mouth can overstretch and cut the hypopharyngeal wall before the teacher discerns the misdirected pressure. The first thing to teach the pupil is that if he puts an esophagoscope into a patient's throat and pushes on it the one place it will not go is into the esophagus." The two most common sites of perforations of the esophagus due to the introduction of the tube itself are the left pyriform sinus and the cardiac portion of the esophagus. Improper position of the patient's head

is frequently responsible for the perforation through the pyriform sinus, as well as the fact that the distal portion of the instrument may be directed too far toward the left side of the pharynx as the attempt is made to pass the esophagoscope through the cricopharyngeal pinchcock. The use of a lumen finder, or small esophageal bougie,[14] serves as a guide to direct the esophagoscope accurately and keep it definitely within the lumen of the esophagus as it is advanced through either of the danger zones mentioned.

Foreign bodies are responsible for a large number of perforations of the esophagus, probably ranking second in frequency to neoplasms as the etiologic factor of such perforations.[15] In infants the signs of perforation may be the first indication that a foreign body has been swallowed, if the foreign body was ingested unobserved (Cases 1 and 2). Similarly, signs of perforation of the esophagus may manifest themselves after the original incident of a foreign body accident has been forgotten, if it produced only minor symptoms when ingested. Consequently, a negative history is valueless and may be misleading. Sharp objects such as safety-pins, rough, irregular bones and dental bridges, will, of course, produce perforations relatively early. Smooth, round objects, on the other hand, such as coins, may produce perforations if they are present over a long period of time and first produce a decubitus ulcer of the esophageal mucosa.

The improper use of forceps through the esophagoscope is another manner in which instruments play a role in esophageal perforation. The insertion of forceps through the musculature of the cricopharyngeus in an effort to grasp a disc foreign body or chicken bone immediately below this mucosal fold results in a perforation which permits infection to pass directly into the retro-esophageal space (Case 4). Similarly, the firm grasp of an irregular object may produce a perforation as the object is pulled upward. Occasionally perforation results from the removal of tissue for biopsy in carcinomatous lesions. This can to some extent be avoided if non-cutting types of forceps are used wherever possible. Ball-bearing forceps which tend to tease tissue from the esophageal lesion rather than actually to cut it are most satisfactory for this purpose. Blind instrumentation is responsible for many more of these surgical accidents than instrumentation under direct vision. The freer use of the esophagoscope as an aid in establishing the diagnosis of esophageal lesions has led to a decrease in this type of accident, but ill-advised attempts at the blind instrumental removal of foreign bodies are still far from uncommon (Case 5).

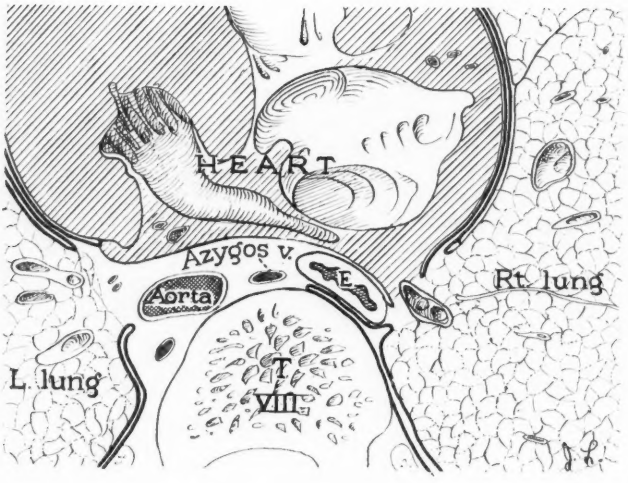


Fig. 3. The position of the esophagus at the level of the eighth thoracic vertebra showing its proximity to the heart and the azygos vein.

Rupture of the esophagus due to external trauma is rare.[16, 17] Aldrich and Anspach[18] described such an accident following a blow on the abdomen. In this instance two large perforations in the esophagus above the cardia permitted free communication between the esophagus and the pleural cavity, with a resultant pyopneumothorax. The child died within 48 hours. Vinson[19] mentions the spontaneous rupture of the esophagus following prolonged violent vomiting, and in another case during the strain of lifting a heavy piece of furniture. The same author reports another case of rupture of the esophagus[20] occurring as the result of an automobile accident, the patient's chest striking the steering wheel. In this case, the perforation extended into the trachea and bronchi as well as into the mediastinum. The patient died as the result of a sudden, exsanguinating hemorrhage.

Caustics which are swallowed either in suicidal attempt or by accident may produce an esophageal perforation by simple cauterization and erosion of the esophageal walls. Such cauterization is usually associated with widespread necrosis, frequently involving the stomach. Blind bouginage used in the treatment of lye burns or

other esophageal stenoses is also responsible for a large number of esophageal perforations. This is due to the fact that strictures of the esophagus are never symmetrical and the lumen is eccentric and frequently surrounded by edematous mucosa (Case 6). The tendency for bougies to pass into a pocket next to the lumen rather than finding their way through the lumen is a fairly common one. Tears due to operative dilatation which is too vigorous occur not at the point of the stricture but between strictures, since the stricture itself is made up of dense, tough tissue.

SIGNS AND SYMPTOMS OF ESOPHAGEAL PERFORATION

Signs and symptoms of esophageal perforation depend upon the site of the perforation and the time interval between the perforation and the examination. The etiological factor likewise determines to a large degree the type of pathology which will be found. The usual symptoms which follow a perforation of the esophagus, whether it be due to a foreign body or to instrumentation in attempting to remove a foreign body, are pain and tenderness which increase with swallowing. This pain may be localized to the point of the perforation or between the shoulders if the lesion is high. In thoracic perforations the patients complain of pain radiating to the back or of a severe substernal pain and excruciating tenderness over the sternum or epigastrium. This may be associated with emphysema extending into the neck, a rapidly rising pulse rate, high temperature, evident shock, and gradually increasing dyspnea. Physical findings and X-ray studies may give evidence of a widening of the mediastinal shadow, and lateral X-rays of the neck reveal a thickening of the postlaryngeal and posttracheal shadow. These signs and symptoms may soon be associated with a severe toxemia. Perforations into the pleura are accompanied by a pneumothorax and empyema whereas perforations into the trachea result in severe attacks of coughing and the expectoration of all swallowed foods and fluids. Occasionally perforations into the great vessels of the neck and thorax produce repeated hematemeses and tarry stools; they may produce an uncontrollable, exsanguinating hemorrhage immediately, however. Clinical indications of perforations into specific organs or spaces will be discussed separately.

TREATMENT—GENERAL CONSIDERATIONS

Prophylaxis must be mentioned in a discussion of the treatment of esophageal perforations because these injuries are so frequently the result of instrumentation. An adequate instrumentarium in trained

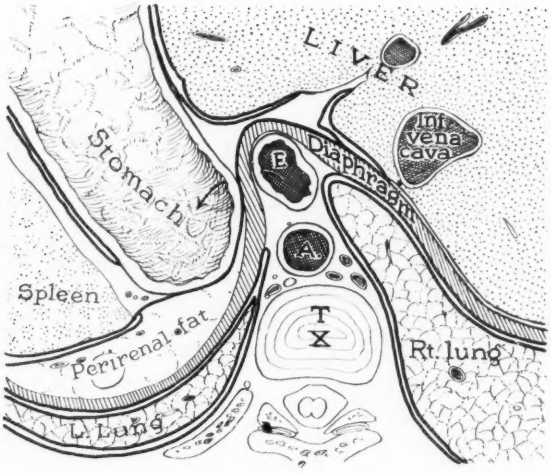


Fig. 4. The relationship of the esophagus at the cardia.

hands is extremely important for the safe management of esophagoscopic problems. Hasty, ill-prepared attempts to remove foreign bodies which require thorough study before the mechanical problem can be solved must be avoided.[21] The use of blind instruments, including coin-catchers, probangs, or even long hemostats, must certainly be considered as obsolete surgical practices in view of the instrumentarium available for direct esophagoscopic procedures. In addition, the assistance of biplane fluoroscopic guidance to the open tube manipulation gives constant information as to the position of parts of the foreign body not actually within the endoscopic field. However, even fluoroscopic assistance is not without an added element of danger because the relation of points on the foreign body with the mucosa cannot be seen under the fluoroscope during the esophagoscopic manipulation. Jackson and Jackson[22] state this in an axiom: "You can visualize the tube and the foreign body; you cannot see what is between them."

Both surgical and non-surgical measures must be considered in the management of the complications of esophageal perforation. It is essential that the esophagus be placed at complete rest, and, in order to do this, the problem of administering food and fluids must be dealt with through the use of intravenous medication, a feeding

tube, or a gastrostomy. The caloric and fluid intake can be kept quite high by means of intravenous medication over a period of several days. However, if the perforation of the esophagus is a large one or the complications exceptionally severe, a gastrostomy will provide the best means of supplying a sufficient caloric and fluid intake. The introduction of a feeding tube, passed through the nose and guided into the cervical esophagus, may lead to grave complications if the edema associated with the perforation prevents the passage of the tube directly into the esophagus and into the stomach.[20] The tube may be guided into the esophagus by direct means, lifting the larynx forward with a laryngoscope if edema associated with the existent pathology prevents its introduction or easy passage through the cervical esophagus. Accurate visualization of the end of the feeding tube is facilitated if the procedure is done under fluoroscopic control. In this manner one may be certain the tube is not advancing through the perforation into the structures adjacent to the esophagus.

Postural drainage in the Trendelenburg position is an exceedingly important adjunct to the treatment of high esophageal perforations. Ochsner and DeBakey[23] stress the prophylactic importance of this therapeutic procedure during operations for mediastinal drainage as well as during the postoperative period.

Chemotherapy likewise must be considered in every case. The advent of sulfanilamide and associated drugs has given a tremendous impetus to chemotherapy. Certainly the use of one of these drugs is indicated in esophageal perforations, depending upon the nature of the organism recovered from the pus. The use of intravenous arsenicals is exceedingly important as well, since infections in this area are bathed with saliva containing spirilla and fusiform organisms. Since these organisms are anaerobic in character, the use of sodium perborate or hydrogen peroxide mouth washes is likewise efficacious. The specific antitoxin for B. welchii or a polyvalent gas bacillus serum may be indicated in certain cases.[24]

SPECIFIC COMPLICATIONS OF ESOPHAGEAL PERFORATIONS

Infections. Infections of the periesophageal tissue spaces are the commonest complications of esophageal perforations. Infections following perforations of the posterior wall of the hypopharynx or laryngopharynx should be included under this category because, while these perforations are not actually through the wall of the esophagus, they occur immediately above the entrance of the esophagus, usually during an esophagoscopy, and enter the same space into which perforations of the cervical esophagus enter. Such perforations may be

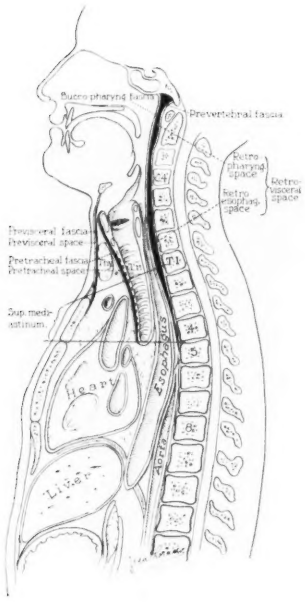


Fig. 5. A diagrammatic midsagittal section showing the potential tissue spaces of the neck and chest and the major fascial planes.

considered clinically as esophageal perforations in the same manner that pulsion diverticulae of the hypopharynx are grouped with diverticulae of the esophagus.

In attempting to classify infections following esophageal perforations one is at once impressed by the number of variable factors upon which the nature of the resulting inflammatory process will depend. Consequently, cases must, to a large degree, be individualized. However, a general classification is possible which is an aid in determining both prognosis and treatment.

In general, infections following esophageal perforations may be divided into two major groups. The first is composed of the localized infections of the periesophageal tissues which may be subdivided into localized periesophagitis and periesophageal abscess. This group is characterized by a mild cellulitis accompanied by a low grade fever, pain, difficulty in swallowing and occasionally by emphysema. There

are usually relatively few systemic symptoms, but the local manifestations may become severe. They are essentially mechanical in nature in their most pronounced state, consisting of marked esophageal or even tracheal obstruction as the abscess becomes walled off and increases in size. Such abscesses may burrow into the great vessels, the trachea, or even into the esophagus, or may produce tracheal compression severe enough to necessitate a tracheotomy if the abscess cannot be adequately drained. The number of instances in which the mildest of the infections in this group heal spontaneously is of definite significance.

The second major group is made up of the fulminant, extensive, overwhelmingly septic cases of mediastinitis which follow esophageal perforation. They are the result of direct introduction of esophageal, oral, or gastric contents into the mediastinum through the wall of the thoracic esophagus, or may be produced by the downward extension of an infection in the visceral space which resulted from a perforation of the cervical esophagus. Such inflammatory processes are usually rapidly progressive and may involve the entire retroesophageal space from the retropharyngeal area to the diaphragm in 24 to 36 hours. The classical signs and symptoms of such inflammatory processes are the acute, obvious toxicity of the patient as evidenced by the prostration, rapid pulse, chills, temperature elevation, and marked leucocytosis. Pain and tenderness in the neck and excruciating pain on swallowing are further symptoms. The patient may have laryngeal symptoms as well, the mounting edema of the soft structures producing hoarseness and dyspnea, which progress to acute laryngeal obstruction in this manner rather than by compression as in the previous group. The rapidity of extension of infections into the mediastinum from perforations of the cervical esophagus is due to the anatomical structure of the fascial spaces. As Head[25] points out, the extreme ease and frequency with which this descent may occur is only rarely noted in infections in other parts of the neck.

As stated above, all gradations of these two principal types of infections may be observed. A third group is frequently added to these two major groups, and includes those phlegmonous infections of the mediastinum which clinically may be considered to be midway between the other groups. These cases are characterized by the fact that both local and systemic symptoms are present but not as marked in either case as in the classes mentioned above.

Treatment. Surgical drainage of the mediastinum is the most important single therapeutic factor in the management of compli-

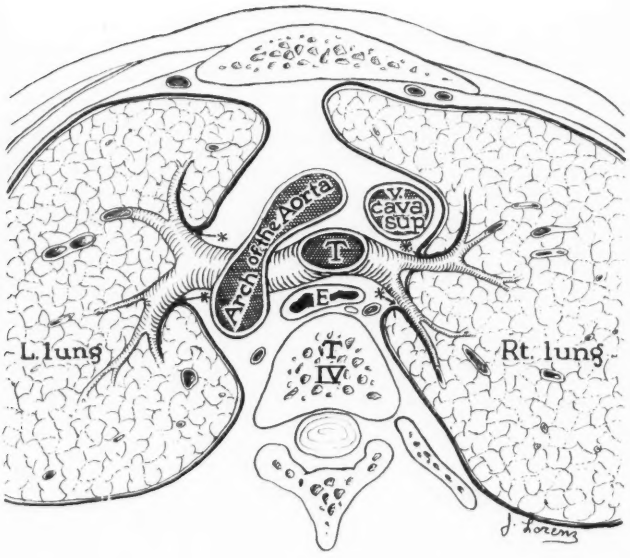


Fig. 6. The position of the esophagus at the bifurcation of the trachea (fourth thoracic vertebra). Note that the para-esophageal space extends along the bronchi at this level as the pleural reflexions at the hilum extend along the bronchi. (*) The mediastinal infections extending into these areas produce the characteristic hilar markings or increase in "root shadows" which are significant in the roentgen interpretation of mediastinitis.

cations of esophageal perforation. The operative approach must be determined by the site of the perforation and by the extent of the inflammatory process which is associated with the perforation. Mediastinitis resulting from perforations of the cervical esophagus may best be drained by a cervical mediastinotomy. According to Head[25] this is effective to the level of the fourth dorsal vertebra. Perforations of the mid-thoracic or cardiac esophagus resulting in mediastinal suppuration may occasionally point upward, advancing through the fascial constriction at the bifurcation of the trachea. In such rare instances the suppurative process may be drained satisfactorily through a cervical mediastinotomy. However, a posterior mediastinotomy is more frequently indicated in the infections associated with perforations of the distal esophagus.

Indications for surgery. Marked variations of opinion in the literature regarding the indications for surgery are surprising. There

is no question that when an inflammatory process is advancing through the retro-esophageal tissues or the mediastinum immediate external drainage is indicated. The high mortality of mediastinitis caused by esophageal perforation is in part due to a failure to adhere to this fundamental surgical principle. The low mortality rate in series of cases in which this surgical procedure is performed as soon as the diagnosis of an esophageal perforation is established is further concrete evidence of its efficacy.

A significantly large group of borderline cases exists, however, that they may be discussed separately. A review of the literature and personal experience indicate that many cases of esophageal perforation recover spontaneously. This is due in part to the fact that when the esophagus is perforated by an instrument or a foreign body the perforation itself provides a route for drainage into the esophagus. This borderline group is made up of the patients on whom an endoscopic procedure has just been completed that was unusually difficult or was originally performed for a difficult foreign body extraction. Such patients may complain of pain on swallowing and tenderness on palpation over the suprasternal notch and sides of the neck and may develop an elevation in temperature to 99 or 100 F. Occasional cases may have an associated slight cervical emphysema. While some[26] recommend surgical drainage at once, others[27] suggest more conservative management, employing all the non-surgical measures such as posture, chemotherapy, absolute elimination of food and fluids by mouth mentioned above. McGibbon and Mather[15] state, from a review of the literature as well as an analysis of their own cases, that subcutaneous emphysema is not in itself an indication for surgery unless there is evidence that it is followed by infection. Frequent temperature readings as well as frequent white blood cell counts are indicated. Should any increase in the severity of symptoms or findings develop, immediate surgery, cervical mediastinotomy, is, of course, imperative.

A cervical mediastinotomy has long been the operation of choice in the treatment of mediastinitis due to esophageal perforation. Von Hacker[28] described the technique of this operation in the treatment of periesophageal and mediastinal phlegmons, and later Marschik and Vogel[29] stressed its importance in the management of perforating foreign bodies. This operation is still the procedure of choice, and modifications of its technique have recently been described by Furstenburg,[5] Ochsner and DeBakey,[23] Heatly and Pearse,[30] and others.

The high incidence of mortality from perforations of the esophagus due to gunshot wounds during the First World War led

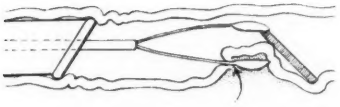


Fig. 7. Perforation of the mucosa of the posterior esophageal wall made in an effort to grasp a coin. A similar perforation into the cricopharyngeal muscle is one of the commonest methods of instrumental esophageal perforation.

Marschik[31] to suggest the so-called prophylactic mediastinotomy in all cases in which a missile had perforated the esophagus. This operation consisted of an immediate cervical mediastinotomy and, if the mediastinum was not already infected, a gauze pack was placed in the mediastinum to act as a barrier to the descent of the infection. The concept of establishing external drainage before an infection developed which might travel downward with extreme rapidity became a very popular one because of the success which followed this type of treatment in the previously fatal gunshot wounds. This operative procedure was apparently applied later as a justification for immediate surgery in any type of esophageal perforation. The current literature is confusing in its interpretation of this operation, and it is unfortunate that the term prophylactic mediastinotomy has received such widespread use, since such a procedure need not be generalized to become indicated in the management of all deep infections of the neck. Lederer and Fishman[32] recently reviewed this problem and used the term to apply to a mediastinotomy which was done relatively soon after the diagnosis of an esophageal perforation was made, the perforation being from within outward. They were divided in their opinion as to the efficacy of this procedure but presented cases which tended toward advocating a conservative type of management. It must be stressed that a cervical mediastinotomy, performed to drain an advancing mediastinal infection is not, in reality, a prophylactic operation. It merely provides drainage for an infected space. On the other hand, the immediate surgical exposure of the mediastinum in cases of foreign body perforation of the cervical esophagus should, it would appear, be avoided because of the danger of opening the tissue spaces of the mediastinum to permit an easy advance of infection or even actually to carry infection into the mediastinum.

Perforations of the Pleura. Perforations of the pleura may occur as complications to esophageal perforations because of the close proximity of the pleura of both lungs to the esophagus. Such perforations carry infection into the pleural cavity or allow air to enter the pleura during the negative intrathoracic pressure of inspiration, to be followed by infection. They are not uncommonly the terminal phase of an esophageal carcinoma and may occur following the ingestion of a caustic. Symptoms are usually those of sudden, acute pain in the affected chest, dyspnea and prostration, and are associated at first with signs of a pneumothorax which rapidly changes to a pyopneumothorax. Aspiration of fluid may reveal food particles, milk, or stomach contents, *B. acidophilus* and yeasts being common organisms found on bacteriologic examination.[33] Treatment consists of the prompt, adequate drainage of the pleural cavity and the placement of the esophagus at rest through use of a feeding tube or gastrostomy. Other supportive measures are described above.

Hemorrhage. Hemorrhage complicating an esophageal perforation is most frequently produced by a foreign body such as a safety-pin which has eroded through the esophageal wall and into the aorta. Tucker[31] describes a fatal hemorrhage from such an accident. In 59 cases in which safety-pins had lodged in the food passages, Clerf[35] describes a different mechanism of vascular erosion in the single fatality of this large series. Death occurred two weeks after the safety-pin was removed, the child apparently having been well, and was due to a massive hemorrhage. Autopsy revealed a small pouch on the anterior wall of the esophagus. The bottom of the sac was eroded and communicated with the innominate artery. There was no evidence of mediastinal suppuration. The author felt that the point of the pin had perforated the esophageal wall in close relation to the innominate artery, injuring the vessel wall. Blood cultures at autopsy revealed hemolytic streptococci. Thus, invasion of the great vessels with a resultant fatal hemorrhage may take place through either the direct puncture of the vessel by an object perforating the esophagus, or it may be due to the erosion of a vessel wall produced by an inflammatory process.

Emphysema. Mediastinal or cervical emphysema is one of the most characteristic signs of esophageal perforation. Air escapes into the soft tissues of the neck or mediastinum due to negative intrathoracic pressure during inspiration, and to some degree indicates the extent to which infectious material may have penetrated the periesophageal tissues. According to Jackson and Jackson,[36] it may prove fatal by pressure or shock even without infection. Such deaths

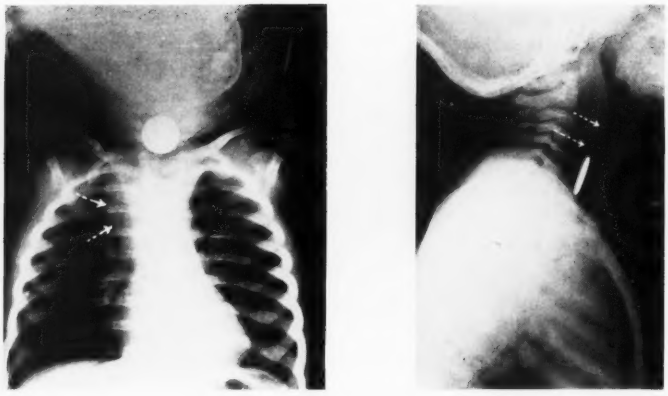


Fig. 8, Case 4. Mediastinitis following unsuccessful attempts to remove a penny from the cervical esophagus of a two-year-old infant. The width of the shadow of the superior mediastinum is apparent on the anteroposterior view. Air-containing spaces may be noted in the retropharyngeal area on the lateral projection.

have been noted with mediastinal emphysema accompanying tracheotomy. However, unless the emphysema is marked or shows a tendency to progress, it requires no treatment *per se*. The roentgen evidence of radiolucent areas in the retropharyngeal infections need not necessarily be due to esophageal perforation but may be due to the presence of gas-forming organisms.[23]

Perforations of the Pericardium and Heart. Perforations of the esophagus involving the pericardium or the heart itself are, in almost every instance, due to foreign bodies. Such perforations are usually relatively slow in their progression of symptoms, an adhesive pericarditis forming about the perforating foreign body. Decker[17] discusses the problem of foreign bodies in the heart and pericardium, reviewing the literature and summarizing the therapy in 109 cases. While the majority of Decker's cases are due to perforations from outside the body, in occasional cases a foreign body migrated through the esophageal wall and into the pericardium. A complete discussion of this complication of esophageal perforations is outside the scope of this paper.

REPORT OF CASES

CASE 1.—B. S., a seven-month-old, white female infant, was seen by Doctor N. T. Welford in extremis a few moments before death. Stimulants were promptly administered, but the infant expired before even the briefest examination could be completed. The history, obtained later, revealed that the infant had been well until approximately two weeks before death. At this time the child was said to have had several mild coughing spells, but this history was elicited only in the face of the autopsy findings. For another week after this the child seemed well again but coughed some more at the end of that time. A cough medicine was prescribed, and the cough seemed to subside. At the end of the second week the child suddenly began to vomit blood and appeared pale and limp. A stimulant was given, and the child momentarily recovered from what appeared to have been a mild shock. However, the following day hematemesis continued, and blood and tarry material appeared in the stools. The child appeared pale and in shock. She died approximately 16 hours after the beginning of hematemesis. A postmortem examination was performed and the relevant findings were the presence of an open safety-pin embedded in large masses of clotted and half-clotted blood in the fundus portion of the stomach. There was a diverticulum of the esophagus with perforation into the mediastinum, and opposite this site there was a perforation of the aorta. There was mediastinal hemorrhage, a diffuse mediastinitis, and mediastinal lymphadenitis.

The case is unusual in its course and complications. It must be assumed that the safety-pin lodged in the esophagus where the open point caused a traumatic diverticulum of the esophagus with perforation, perhaps purely on a traumatic basis, perhaps with the assistance of a developing infection into the mediastinum and into the aorta. It may be assumed that the developing infection was at least auxiliary, bringing about the leakage of blood from the aorta into the mediastinum and into the digestive tract in large masses. Massive hemorrhage and anemia were the immediate causes of death.

CASE 2.—B. J. O., a one-year-old female infant, had been ill four days with a low grade fever and restlessness, and had refused her feedings. Progressive pulmonary findings in the right chest were thought to be due to pneumonia, but these findings changed rapidly on the day prior to admission. Fluoroscopic examination revealed a safety-pin, open, in the esophagus, point to the right, and a right pyopneumothorax. On admission to St. Luke's Hospital the temperature was 102.8 degrees and the respiratory rate 90-104 per minute. The infant was extremely toxic and dehydrated. A chest aspiration by Dr. Willard Van Hazel revealed a Type XIX pneumococcus. Intravenous fluids, transfusions, Type XIX anti-pneumococcus serum, sulfapyridine and other supportive measures were instituted. All food and fluids by mouth were discontinued. The following day a closed drainage system was established for the right chest. The safety-pin was removed one week later, the pin being carried to the stomach, straightened and extracted under fluoroscopic guidance. During this time, any attempt to feed the infant by mouth resulted in the drainage of the food through the empyema drain. Consequently, a gastrostomy was done. Improvement was very slow, but finally, five weeks after admission, the empyema tube could be removed. The infant continued to improve for another week when she suddenly expired following a gastrostomy feeding. Postmortem examination revealed the fact that the esophagus and right chest had completely healed. Death was due to a perforation of the

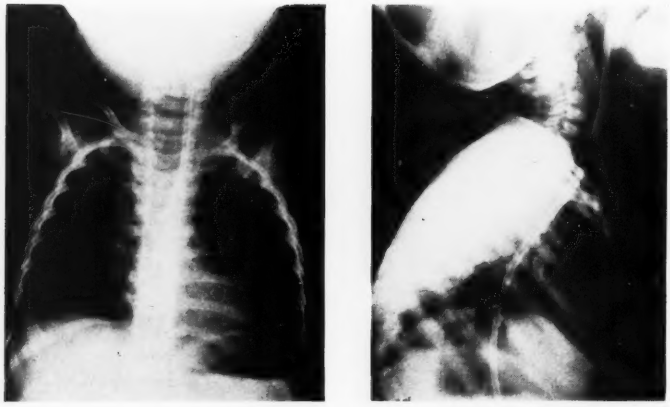


Fig. 9, Case 4. A nasal feeding tube in place following removal of the coin and drainage of the abscess by aspiration directly through the perforation in the posterior wall of the cervical esophagus.

stomach and left diaphragm by a decubitus ulcer produced by the gastrostomy tube, the contents of the stomach flooding the left thorax.

In this case a complete perforation of the esophagus led into the right pleural cavity to produce a pyopneumothorax. Adequate surgical drainage, with esophageal rest made possible by a gastrostomy, eventually resulted in a healing of the perforation. Death occurred after the perforation had completely healed and was only indirectly due to the perforation.

Case 3.—R. B., an eight-month-old male infant, was admitted to St. Luke's Hospital five days after having swallowed an open safety-pin. The infant had had a non-productive cough and a temperature varying between 102° and 103° F., and had had a bloody stool. X-rays revealed an open safety-pin in the thoracic esophagus, point to the left. There was definite hilar thickening. An esophagoscopic examination was made shortly after admission, and the pharynx and upper esophagus were found to contain a small amount of dark blood. The safety-pin was seen covered with reddish-brown blood clots, and the surrounding mucosa was ulcerated and freely bleeding. The entire point branch was found to be embedded in the mucosal wall. The keeper was grasped and the esophagoscope advanced over it as the pin was carried to the stomach, straightened and extracted. Fluids were withheld by mouth and supportive measures instituted. The temperature gradually subsided, and the chest picture steadily cleared. Convalescence was uneventful.

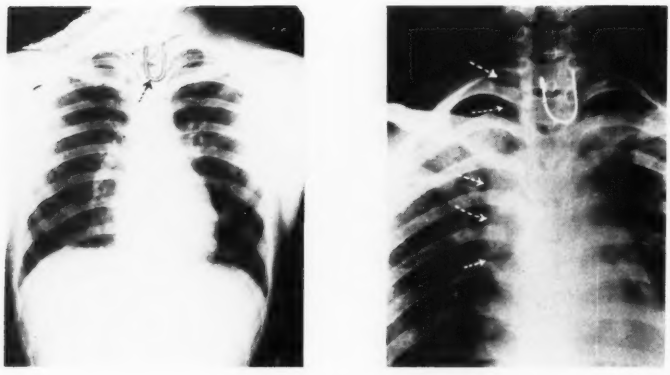
It can, of course, be questioned that this was a true esophageal perforation. A laceration of the esophageal wall might conceivably produce blood in the esophagus and the tarry stool, but from the endoscopic appearance there could be no question that the point branch had completely perforated the esophagus.

CASE 4.—S. H., a two-year-old, female infant, swallowed a penny on April 3, 1937. Three attempts were made to remove the coin, all under ether anesthesia, on April 5, 6 and 9. The second and third attempts were followed by fever of 105.8°. The child was admitted to the Children's Memorial Hospital April 10, 1937, and an X-ray showed the penny still present in the cervical esophagus. The pharynx, larynx, and upper thoracic esophagus were pushed forward, and the upper mediastinal shadow, as far as the tracheal bifurcation, was wide and fuzzy. The retropharyngeal and retrolaryngeal tissues contained air spaces. The pharynx and cervical esophagus were examined with an esophageal speculum on April 11, 1937, and the coin removed. The posterior wall of the cervical esophagus was found perforated, and thick pus exuded through the perforation. The perforation was fairly large and was easily aspirated repeatedly with a laryngeal aspirator. A nasal feeding tube was inserted, being guided past the perforation under direct vision. Drainage continued through the perforation, and this was aided by frequent pharyngeal aspirations with nasal catheter suction. There was a gradual return to normal of the retrolaryngeal soft tissues and the patient made a complete recovery.

From the endoscopic picture of this case, it was quite obvious that a perforation had been made through the posterior wall of the cervical esophagus by the lower blade of an endoscopic forceps. X-rays on admission to the Children's Memorial Hospital demonstrated the presence of an abscess extending from the base of the skull to the fourth thoracic vertebra, with air in the retro-esophageal tissues. The copious discharge of frank pus through the perforation from the retro-esophageal tissues indicated the adequacy of this drainage route and external drainage was postponed. As the general condition slowly improved the retrotracheal shadow gradually returned to normal, and it became evident that external drainage would be unnecessary.

CASE 5.—D. W., a 14-year-old boy, was admitted to St. Luke's Hospital with the history that he had swallowed a curtain hook during the previous evening while reading. The roentgen examination demonstrated the hook to be in the cervical esophagus, point upward. Blind attempts at removal had been made elsewhere under general anesthesia with hemostats. The attempts were discontinued after one and a half hours when a bilateral cervical emphysema was elicited by palpation and visualized by X-ray. The X-ray showed in addition a markedly widened mediastinal shadow and demonstrated that the hook was further laterally and higher than on the first film. Because of the apparent esophageal perforation, further instrumentation seemed contra-indicated. He vomited frequently during recovery from the anesthetic. His temperature was 100.4° and the white blood cell count, 10,500. On admission to St. Luke's Hospital he was placed in a Trendelenburg position, given a sodium perborate mouth wash and powdered bismuth subnitrate on the tongue. All foods and liquids by mouth were withheld, and fluids administered intravenously. During the following five days he was kept in bed, remaining in the Trendelenburg position. His temperature (rectal) only once rose to 101°, and the white blood cell count remained below the count on admission. At the end of this time his temperature and white count were normal, and roentgenograms of his entire body failed to reveal the foreign body. Liquids were swallowed with ease and without any pain whatsoever. His subsequent course was uneventful.

In reconstructing this case, it is apparent that during the blind attempts to remove the hook the hook was grasped and pulled upward, embedding its pointed



A *B*

Fig. 10, Case 5. A curtain hook in the cervical esophagus of a 14-year-old boy. The original film *(A)* shows the hook with its point upward and toward the left. The second film *(B)* was made one and a half hours later following blind attempts at removal. Cervical emphysema and a widening of the mediastinal shadow are noted.

end. During the time the patient was in the hospital, the stools were carefully examined but the foreign body was not recovered. It is possible that the hook became dislodged between the time the second X-ray was taken and his admission to the hospital, because it was during this time that he vomited while recovering from the anesthetic.

Case 6.—Z. S., a one-year-old, colored, female infant, had swallowed lye two hours before admission to the Children's Memorial Hospital on July 25, 1940. The burn in the mouth was exceptionally severe, and the usual antidotes were administered. Breathing became increasingly difficult through the day, and it became necessary to tracheotomize the child about eight hours after admission. Finally, a gastrostomy was performed by Dr. Montgomery on August 3, 1940, the child previously having been unable to take any fluids by mouth since the ingestion of the lye. Six weeks later direct examination of the larynx revealed a marked distortion of all laryngeal structures and a complete absence of the epiglottis. Small bougies were passed gently into the esophagus, but these met with resistance in the distal third. The child had been unable to swallow a string. Following this examination and dilatation, the child regurgitated a gastrostomy feeding through the mouth. Prior to this, nothing had been swallowed or regurgitated. Her temperature rose rapidly, and the tissues of the neck became emphysematous. Physical findings and X-rays demonstrated a right hydropneumothorox. Closed drainage was promptly established by Dr. Montgomery, and stomach contents were recovered from the aspirated contents of the chest. The child's subsequent course was stormy, and she expired 23 days later. Postmortem examination revealed an extensive destruction of the hypopharynx and esophagus, with two perforations

of the esophagus at the level of the tracheal bifurcation. Stomach contents were found to have recently entered the left pleural space. A generalized fibrinopurulent peritonitis was found in addition to the empyema of the right pleural space.

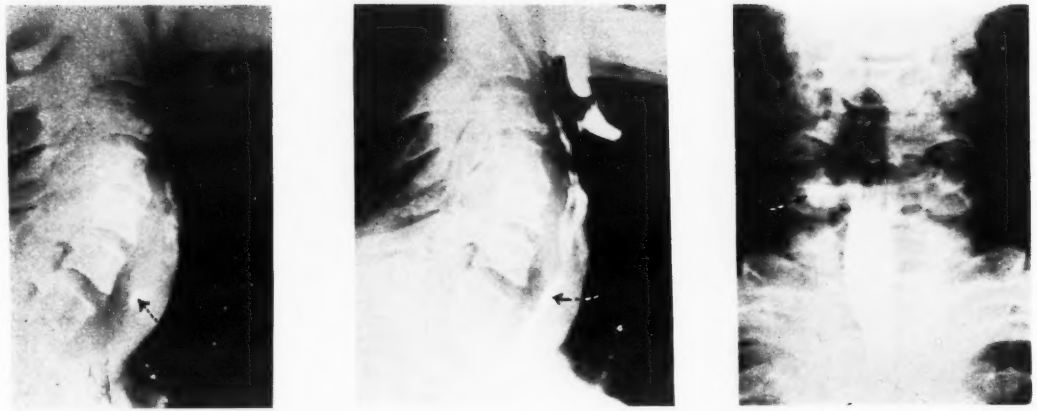
This child had an unusually extensive lye burn, necessitating both gastrostomy and tracheotomy. While it is difficult to trace the actual route through which the infection traveled to reach the peritoneum because of the long time interval between the perforation of the esophagus and death, it apparently extended posteriorly and inferiorly, passing through the diaphragm at the esophageal hiatus.

CASE 7.—L. G., a 71-year-old, white female, swallowed a chicken bone one week before admission to the Research and Educational Hospital. Two previous attempts at removal and one shortly after admission were unsuccessful, in spite of apparent positive X-ray evidence of a bone in the cervical esophagus. Fluoroscopic studies and esophageal films with barium, however, demonstrated that the bone was in the soft tissues of the neck, lying in a pocket which was still connected to the esophagus. The leucocyte count at this time was 12,500 and the temperature, 99.4°. A surgical exploration was made through an incision along the anterior border of the right sterno-mastoid muscle by Dr. Warren Cole, and the site of the perforation exposed. An area of dense inflammatory tissue was found adjacent to the esophagus at this point. In order to avoid breaking this down unnecessarily, an esophagoscope was passed to the ulcerated area seen at this level in the previous esophagoscopy. At this time the bone was found lying partially within the esophageal lumen, and was extracted through the esophagoscope. Thus the surgical manipulation had allowed the bone to re-enter the esophageal lumen, and removal perorally eliminated the necessity of breaking down the inflammatory adhesions which had encapsulated the foreign body. The patient made an uneventful recovery.

This case illustrates the cooperation between surgeon and endoscopist which may be of great advantage in the management of perforating foreign bodies. Such teamwork is advantageous in the surgical extirpation of hypopharyngeal pulsion diverticulae and in many clinics is routinely used in that operation. The significance of this case in this series is that in the surgical procedure the mediastinal space was not opened

CONCLUSIONS

Perforations of the esophagus resulting in perforations of the pleura, trachea or bronchi, or in hemorrhage are as a rule readily recognized and treated according to certain well-established principles. Obvious mediastinal infection demands prompt drainage. This includes not only cases in which there is definite evidence of a mediastinal abscess, but also those cases of gross perforation demonstrated at endoscopy or by X-rays. In a few cases this is established through the site of the perforation itself. Cases 4 and 7 illustrate this point clearly. However, external drainage is, as a rule, far superior to endoscopic drainage.



A B C

Fig. 11, Case 6. *A*. Lateral X-ray of the neck of a 71-year-old patient who had swallowed a chicken bone one week previously. The bone, apparently in the cervical esophagus, was not found during the esophagoscopic examinations. *B*. Lateral view following administration of barium. The bone definitely appears to be in the column of barium. *C*. Anteroposterior view following a swallow of barium. The bone is seen entirely outside the barium column but connected to it by a fistulous tract.

Perforations of the esophagus resulting in inflammatory processes in the tissues surrounding the esophagus are frequent subjects for discussion and there is a wide variation of opinion in the literature as to their management. Case reports of numerous authors as well as those presented herein indicate that many esophageal perforations resulting simply in periesophagitis heal spontaneously. The difficulty in actually establishing the diagnosis of an esophageal perforation and in differentiating between a laceration and a perforation permits a certain conservatism in the management of borderline cases. Questionable cases must be observed frequently by physical examination, chest X-ray studies and routine leucocyte counts. Any increase in findings or symptoms demands prompt, adequate surgical interference.

SUMMARY

1. A brief review of the anatomy of the esophagus and the surrounding structures is presented.

2. The etiology of esophageal perforations and their complications is discussed.

3. The complications of esophageal perforations are:
   a. Infections: periesophageal and abscess.
   b. Hemorrhage.
   c. Perforations of the pleura.
   d. Emphysema.
   e. Pericarditis.
   f. Perforations of the trachea and bronchi.

4. The diagnosis and treatment of these complications are discussed.

5. Representative cases are presented.

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XLIX

ADENOCARCINOMA OF THE LARYNX

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Of all the tumors which involve the larynx, none have received less consideration than adenocarcinomas; yet these growths offer perplexing problems in diagnosis and are among the most serious neoplasms affecting the larynx. Largely responsible for the lack of familiarity with these tumors is their infrequent occurrence; the fact that of approximately 1300 malignant laryngeal tumors seen at the Mayo Clinic in the past 30 years only six were adenocarcinomas is convincing evidence of their extreme rarity. Foremost among the difficulties which beset the laryngologist in the diagnosis of adenocarcinoma are a lack of distinctive symptoms produced by such growths and a want of distinguishable characteristics in their gross appearance. As a matter of fact, on analysis of the majority of laryngeal adenocarcinomas, one finds that they tend rather to resemble more or less completely certain benign lesions. Even when an adenocarcinoma of the larynx is of large dimensions, without microscopic confirmation the clinical recognition of such a tumor is never more than a conjecture, and this is especially true when the growth is encountered in the early stages of its development, a time at which treatment would be most effective.

The adenocarcinomas most frequently encountered in the larynx are low-grade, slow-growing neoplasms, usually of the mixed tumor type. While not true mixed tumors, they possess some of the histologic features peculiar to a mixed tumor. On microscopic examination epithelial-lined spaces are to be found surrounded by a hyaline-like stroma; some of the epithelial cells present malignant characteristics (Fig. 1). In five of our series of six cases, the adenocarcinomas were classified histologically as adenocarcinomas of mixed tumor type, and in two of these cases the neoplasms had cylindroma-like attributes. In all five cases the neoplasms were of a low grade; in three cases they were graded I on a basis of four. In the sixth case the lesion was a highly malignant neoplasm diagnosed as Grade IV mixed

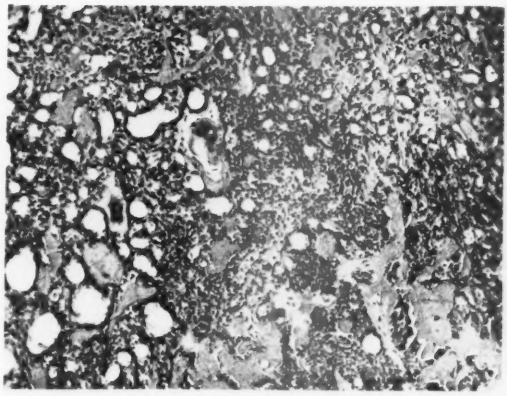

Fig. 1. Adenocarcinoma, Grade I, of larynx (mixed tumor type, X95).

Fig. 2. Lateral roentgenogram of larynx showing a large adenocarcinoma simulating a chondroma. This tumor involved the right side of the larynx.

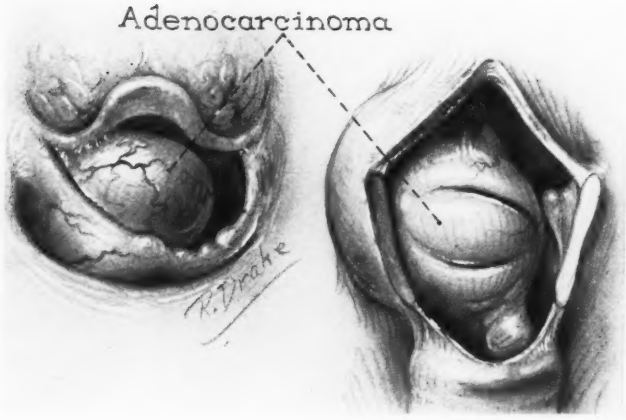

Fig. 3. Large adenocarcinoma of the right side of the larynx. This tumor was situated deep in the lateral wall, and bulged the true and false cords into the laryngeal lumen; *left*, view of tumor by indirect laryngoscopy; *right*, view of tumor through a thyrotomy incision.

squamous-cell and adenocarcinoma. In view of this dual character, there is some question in our minds whether or not this tumor should be included in this consideration of ordinary adenocarcinoma of the larynx.

The majority of laryngeal adenocarcinomas develop very insidiously, which is in keeping with their low degree of malignancy. Further evidence of their tardy growth lies in the fact that in our series of cases the patients, while conscious of some laryngeal disturbance, waited from one to nine years before seeking medical attention. The prominent symptoms produced by these tumors are in the order of their frequency: dyspnea, hoarseness, cough, a sense of fullness in the throat, and, rarely, dysphagia. Although in the early stages of an adenocarcinoma the patient may be but vaguely aware of the laryngeal growth, there is an increasing severity of one or more of the foregoing symptoms in response to the gradual development and extension of the tumor. In contrast to squamous-cell epitheliomas which usually arise on a vocal cord and produce hoarseness, adenocarcinomas seldom primarily involve the vocal cords and are inclined to cause dyspnea as an initial symptom by obstructing the larynx. Of course, if the tumor becomes sufficiently large to displace or to interfere

Fig. 4. Roentgenographic area of increased density due to adenocarcinoma, Grade I, of the subglottic region. This tumor simulates a chondroma; *a*, lateral roentgenogram of the larynx; *b*, anteroposterior tomographic study of the same larynx.

with the function of a vocal cord, hoarseness becomes a prominent symptom. An adenocarcinoma may reach conspicuous dimensions before the patient is aware of dyspnea. The explanation of this fact probably lies in the extremely slow growth of the neoplasm which increases so gradually in size that the patient, being accustomed to the slowly diminishing volume of air, is not conscious of the obstruction until it becomes marked. Furthermore, adenocarcinomas of subglottic origin may involve the extralaryngeal tissues by extension through the thyrocricoid membrane before producing noticeable subjective laryngeal symptoms.

In our experience, adenocarcinomas have a tendency to arise in or near the subglottic region. With the exception of the mixed squamous-cell and adenocarcinoma which was confined to the right aryepiglottic fold, the remaining five tumors in our series all originated in, or involved, the subglottic region. In three cases the neoplasm was limited entirely to the subglottic region; in one case the neoplasm occurred in the right side of the larynx, bulged the vocal cord into the laryngeal lumen and extended both into the supraglottic and subglottic regions. In the fifth case the tumor had been surgi-

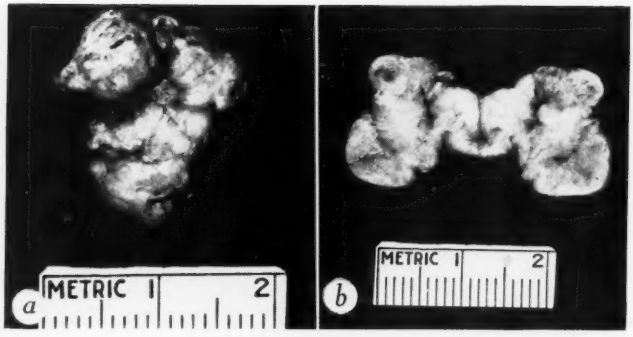

Fig. 5. Adenocarcinoma, Grade I, removed from larynx (same tumor as shown in Fig. 6); *a*, gross specimen; *b*, cut surface of neoplasm.

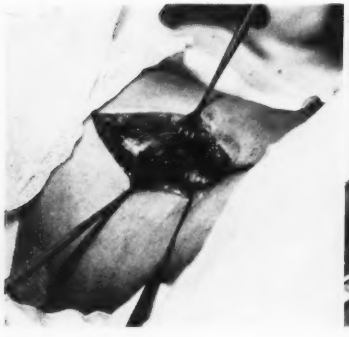

Fig. 6.

Fig. 7.

Fig. 6. Adenocarcinoma, Grade I, of the larynx attached in the right subglottic region. The tumor is exposed through a thyrotomy incision, the chin being to the left. (The tumor is directly above the upper end of the right lower hook retractor.)

Fig. 7. Postoperative photograph of the same lesion as shown in Fig.5; the adenocarcinoma has been removed and its base lightly electrocoagulated. The burned surface can be seen through the thyrotomy incision and lies directly above the upper end of the right lower hook retractor (the chin is to the left).

cally removed and a course of roentgen therapy had been employed before the patient came to the clinic for consideration. Examination at the clinic revealed that the larynx was completely obstructed by edematous mucosa secondary to perichondritis; consequently, it was impossible to determine the nature or site of the lesion. However, tissue removed for biopsy from the subglottic region and obtained through an opening in the trachea revealed adenocarcinoma.

Adenocarcinomas in the larynx appear as firm, smooth thickenings or, more commonly, as lobular or nodular tumors, and are usually not ulcerated. They are covered with normal mucous membrane and not infrequently have dilated vessels coursing over their surfaces. Not only in appearance but also in site do these neoplasms simulate chondromas and amyloid or inflammatory tumors. At least, this was true in our series of cases of adenocarcinoma. There was but one exception: the high-grade, mixed squamous-cell and adenocarcinoma which was a pedunculated, dark reddish tumor resembling an angioma or melano-epithelioma. The appearances of the other five lesions were as follows: The first appeared as a thickening and involved the entire circumference of the subglottic region; this growth was identical in gross appearance to an amyloid tumor. The second was a smooth, rounded tumor attached anteriorly in the subglottic region, and extended anteriorily into the neck just to the left of the median line. As it was a hard mass, a roentgenogram of the laryngeal region was obtained; this revealed an area of increased density similar to a chondroma (Fig. 2). Had it not been for the mass in the neck, which was suggestive of a low-grade malignant tumor, this growth would have been diagnosed clinically as a definite chondroma prior to obtaining a specimen for biopsy. The third tumor was situated deep in the right side of the larynx and bulged the vocal cord mesially (Fig. 3); in view of a nine-year history, this growth was thought to be some form of benign tumor, probably inflammatory. The fourth tumor could not be diagnosed merely by inspection as a course of roentgen therapy had been given a short time before the patient came to the clinic. Since the fifth tumor is to be considered in detail, it is sufficient to mention here that it was a subglottic neoplasm resembling a chondroma.

We would like to stress at this point the importance of obtaining roentgenologic and tomographic studies in all cases in which hard or firm tumors affect the larynx. Although chondromas are the most notable examples of radiopaque laryngeal neoplasms, it is to be remembered that other tumors also at times will present a roentgenologic picture similar to chondromas; this is particularly true of some

tuberculomas, amyloid and inflammatory tumors, and also of adenocarcinomas. Consequently, when a tumor produces a definite area of increased density on a roentgenogram, one should not conclude immediately that the growth is cartilaginous in character.

In general, the prognosis in cases of laryngeal adenocarcinoma should be guarded. Were it possible for the patients to be examined early in the course of the disease, the treatment could be carried out most effectively. However, as previously described, owing to the site and slow growth of the majority of these tumors, the patient is often unaware of the condition until it is far advanced. In the case in which the neoplasm was Grade IV, as might be expected the patient died of generalized metastasis two years postoperatively. Of the other five patients who had low-grade adenocarcinomas, the first refused treatment and died one year later. The second had a tumor that had extended into the neck; this growth was inoperable and the patient died two years after his examination. The third patient, who had laryngeal perichondritis secondary to roentgen therapy, is now living and well three and a half years postoperatively; in this instance, laryngectomy was necessary to effect a cure. In the fourth case a large metastatic mass developed at the base of the tongue following removal of the primary growth and caused the patient's death. The adenocarcinoma involving the larynx of the sixth patient was removed by thyrotomy, as will be described later; this patient is now well, although only six months have elapsed since her operation.

We believe that these cases serve to emphasize the serious problems which arise in the care of laryngeal adenocarcinomas. In the majority of instances, the treatment should consist of surgical procedures of a radical nature. If the lesion is not too extensive, it can be removed successfully by thyrotomy (laryngofissure) in which the growth is excised and its base thoroughly destroyed with surgical diathermy. However, when a considerable portion of the larynx is involved by the tumor, laryngectomy becomes essential. Owing to the site and seriousness of the majority of such growths, one should not rely upon intralaryngeal methods (direct or suspension laryngoscopy) to effect a cure. Unless the neoplasm is highly malignant, irradiation is of little or no value in the treatment except as a palliative measure in cases in which the lesion is hopelessly inoperable.

Although radical surgical measures are of the utmost therapeutic importance, if there are to be no failures in the management of laryngeal adenocarcinomas emphasis must be placed upon the problem of early diagnosis. Unless these tumors can be recognized at a

stage in which the lesion is still confined within the cartilaginous limits of the larynx, no surgical technic can be expected to produce entirely satisfactory results. Unfortunately, an early diagnosis is beset with many difficulties. Not only do countless patients delay seeking medical attention, but, as has been pointed out, when an adenocarcinoma is encountered, even in an advanced stage, there are no physical characteristics by which it may be identified. Consequently, conclusiveness of the diagnosis is based not upon laryngoscopic inspection of the tumor but upon its histologic appearance. Since no clinical method has been established by which adenocarcinomas may be distinguished with certainty other than by biopsy, it is obvious that the early recognition of these neoplasms is dependent upon the microscopic examination of every tumor involving the larynx. In the diagnosis and management of all laryngeal growths, a biopsy can do no harm and in the case of adenocarcinomas it is invaluable.

We wish to report one case in detail because it exemplifies a typical laryngeal adenocarcinoma, typical not only in the symptoms occasioned by the presence of this growth in the larynx but also in the external appearance of the lesion. This tumor was seen at a stage in which, by proper treatment, one could expect with a high degree of assurance to effect a permanent cure. Moreover, this case illustrates the importance of a microscopic examination in the diagnosis of all laryngeal tumors.

REPORT OF A CASE

A woman, aged 51 years, came to the clinic complaining of a sensation of pressure in the throat, a persistent cough, and increasing shortness of breath. Although she had been conscious of these symptoms for one year, the dyspnea had become considerably worse one month prior to her registration at the clinic and had induced her to seek medical attention.

Examination of her throat by indirect laryngoscopy disclosed a subglottic tumor apparently attached to the posterior surface of the cricoid cartilage. The tumor was covered with normal mucous membrane and obstructed much of the laryngeal lumen. The vocal cords performed normally during phonation. Roentgenologic and tomographic examination revealed a nodular tumor which was about 2 cm. in diameter and arose in the right posterior aspect of the subglottic region (Fig. 4a and b). Because of the laryngoscopic findings and in view of the roentgenographic appearance, the neoplasm was thought to be most likely a chondroma. However, we were aware of the fact that only by means of a biopsy could the diagnosis be assured. Before attempting to perform a biopsy or to manipulate the larynx in any manner, it was considered necessary to perform tracheotomy for the relief of dyspnea.

Accordingly, following a general examination in which nothing of any consequence was discovered, the trachea was opened just below the cricoid cartilage.

Through this opening the subglottic tumor could be visualized directly and clearly. A small piece of the growth was excised for microscopic examination. The neoplasm was reported to be an adenocarcinoma, Grade I, of mixed tumor type (Fig. 1). Since this neoplasm was of low grade and was well localized, we believed that it could be effectively removed by thyrotomy. However, in cases in which laryngeal obstruction has been present for any length of time, we believe that the operative risk is appreciably reduced by postponing the laryngeal operation until the patient is completely free of tracheal irritation secondary to tracheotomy. Consequently, in this case thyrotomy was delayed for three weeks.

Thyrotomy was carried out with no difficulties; the thyroid and cricoid cartilages were divided anteriorly with an electrically driven circular saw. On exposure of the interior of the larynx, the tumor was found to be nodular, about 2 cm. in diameter, and attached by a broad base in the right posterior aspect of the subglottic region (Figs. 5 and 6). This growth was excised and, since it was encapsulated, the bed of tissue from which it had been removed was only lightly electrocoagulated (Fig. 7). A split rubber tube drain was inserted, and the wound was closed by means of subcutaneous catgut sutures and silk skin sutures.

The patient made an uneventful recovery and was dismissed from the hospital on the fourteenth day after the operation. On the following day, the swelling within the larynx had subsided to a degree that the tracheal cannula could be removed. The patient was dismissed from our care in another five days. Six months have elapsed since this operation, and there is no sign of a recurrence so far. Although six months is entirely too short a time to rule out the possibility of recurrence, in view of the character and site of this adenocarcinoma, a recurrence is certainly not to be anticipated.

MAYO CLINIC.

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CHONDROMA OF THE LARYNX*

Report of Case

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Connective tissue tumors of the larynx are not common. Chondroma is rare. A careful search of the literature reveals that throughout the years only 82 cases of chondroma of the larynx, including the one herein reported, have been placed in medical literature. Of these 82 cases of chondroma of the larynx, 62 are cited by Moore in his article, which is accompanied by an extensive bibliography.

To go into detail as to origin, classification, case histories, etc., would be only a recapitulation of the cases already reported. A complete bibliography is appended to this report.

ETIOLOGY

Virchow stated that this neoplasm takes origin from normal perichondrium, or from the body of the cartilage; cartilaginous "rest" serving as the matrix. These growths may take origin from any of the cartilages of the larynx. One of the features of the chondroma is that they are very slow growing, and range in size from that of a pea to such an extent as to close off the larynx completely, and encroach markedly on the hypopharynx interfering with deglutition. No enlightenment as to the cause of chondroma of the larynx has been brought out in the literature. It seems to occur mostly in the male, and is most common in middle life.

CLASSIFICATION

Muller (Berlin) in 1936 was the first to give these growths their true histological classification. From a practical standpoint the classification of these tumors by Tobeck is perhaps the best, i. e. (a) those chondromata arising on the inner surface of the larynx, and (b) those chondromata arising on the outer surface of the larynx. The case

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herein reported, from a macroscopical appearance, seems to conform to the latter group, that of arising on the outer surface of the larynx, and this type is extremely rare.

SYMPTOMS

These tumors grow slowly, and the symptoms develop gradually but constantly, increasing in severity. Symptoms referable to respiration and deglutition are the most prominent, depending on the original site of origin. The first sign which may cause the patient to consult a physician is a sense of fullness with a small lump on the side of the neck. This small tumor might be noticeable on the side of the neck for years before definite signs of embarrassment, either in respiration or swallowing, manifest themselves. Those tumors arising within the larynx naturally will produce more symptoms referable to respiration than those arising on the outside. Hoarseness appears early and would naturally be an outstanding symptom, or if not hoarseness, then possibly a change in voice, from a slight alteration to a complete aphonia and later dyspnea. The hoarseness and dyspnea depend on the location and size of the tumor, as for instance, those arising on the epiglottis or thyroid cartilage, externally or subglottic, would not at first produce any change in voice. Cough develops and may be pronounced, according to the site and development of the growth. Those tumors arising on the outside of the larynx would present more symptoms referable to deglutition, such as dysphagia, without pain due to the tumor growing into the food passage, until such time as the growth so increases in size as to cause concern to respiration. Then again a palpable mass in the neck may be the first sign, as occurred in the case herein reported.

All the symptoms mentioned above progress slowly until marked laryngeal or esophageal obstruction causes the patient to become really cognizant of the seriousness of the condition.

DIAGNOSIS

It is superfluous to bring out any difficulties in diagnosis other than that these tumors are rare, more apt to be subglottic, and that they may simulate malignancy; this may offer some difficulty. However, if one follows out the usual diagnostic procedure used in arriving at a diagnosis of malignancy, a positive diagnosis can usually be made. With alteration of voice, one would naturally make a laryngoscopic examination, in which he would find, depending on the location of the growth, a narrowing of the glottic chink, or if the growth is

within the larynx, the growth itself. There may be ulceration, which might lead one to suspect malignancy, but after a Wassermann has been taken and found negative, with X-rays of the larynx, as well as one of the chest, and then a histological examination of the biopsy, the condition should readily be diagnosed. A subglottic growth might be entirely overlooked unless a direct laryngological examination is made.

If the growth is on the outside of the larynx, by palpation of the neck one would find the growth attached to the larynx; the growth is hard and may be nodular, and with the history of a small tumor gradually but persistently enlarging, interfering both with deglutition as well as respiration. With these physical and laryngological findings, plus roentgen ray studies and biopsy, there should be sufficient evidence to justify the diagnosis of chondroma of the larynx. A differential point in the diagnosis is that a carcinoma of the larynx may be confused with chondroma, as occurred in one of my cases in which all the histological examinations were negative. The larynx on palpation was of stony hardness, which was of long duration. A diagnosis of chondroma was made, but not until the larynx was completely removed was the final diagnosis made of carcinoma of the larynx.

The following is a brief outline of that case to show how easily chondroma of the larynx may be mistaken for carcinoma.

E. Q., female, aged 40 years, consulted me on November 20, 1934, because of marked difficulty in breathing and swallowing, accompanied by cough and hoarseness. This had been going on for three and one-half years, the first symptoms developing at the time of her first pregnancy. Notwithstanding a family and personal history of tuberculosis, X-rays of the chest and repeated examinations of the sputum were negative. On account of the marked dyspnea at time of consultation, a tracheotomy was immediately performed. A few days later a direct laryngoscopy was done, which showed some edema of the larynx and a paralysis of the left vocal cord; a specimen was taken for histological study, which was reported on as follows: "Shows a thinning out of the surface stratified squamous epithelium. There is an increased amount of loose fibrous tissue and of the mucous glands. Chronic productive laryngitis. No evidence of malignancy." On three other occasions, March 13, 1935, July 10, 1935 and March 25, 1936, further specimens were removed, and in the last report a diagnosis of chronic ulcerative laryngitis was made (possibly of luetic involvement). At this point I wish to say that repeated Wassermann tests were made, all of which were negative. On July 16, 1936 (almost two years later) I requested another consultation, and I quote herewith from the consultant's letter: "I believe this is a chondroma of the larynx. It is impossible to carve out a new larynx from this mass of chondroma, therefore a total laryngectomy is the only procedure." On September 12, 1936, an extensive operation was performed. The larynx, the hyoid bone and practically all of the cervical esophagus along with the thyroid gland were removed en masse. This entire mass was

Fig. 1. Showing specimen of the larynx, thyroid gland, hyoid bone and part of the cervical esophagus removed en masse. Diagnosis: epidermoid carcinoma.

Fig. 2. X-ray photograph showing extent of the chondroma.

subjected to histological study, and the report was as follows: "There is an irregular growth around the cords and an extensive firm growth posterior to the larynx. In the growth surrounding the cords the cartilage is hypertrophied but otherwise normal. Beneath this, embedded in fibrous tissue, there are alveolar epithelial groups. The section from the growth posterior to the larynx shows an extensive fibrous tissue production and diffuse invasion of epithelial groups. The cells are not large, but vary somewhat in size; contain nuclei, which are hyperchromatic and mitotic. There is considerable patchy leucocytic infiltration, chiefly lymphocytes, but no tubercles or foreign body giant cells are present. The diagnosis—epidermoid carcinoma of the larynx." In this case it was necessary to make a new cervical esophagus. The patient died two years later of a recurrence around the tracheal opening, her death occurring eight years after the first symptoms developed.

The above case is cited to show how these two conditions may be confused.

TREATMENT

This naturally depends on the size and location of the growth. A small growth on the inner surface of the larynx can be removed by suspension laryngoscopy or thyrotomy; this has been successfully accomplished in some of the cases reported. When the growth is large and arises from the external surface of the larynx, which not only interferes with respiration, but deglutition as well, nothing short of a laryngectomy will do, and in doing a laryngectomy there is less likelihood of a recurrence when the entire mass is removed than in following a more conservative method of attempting to enucleate the chondroma.

Mr. B., aged 66 years, white, well-developed male, born in Hungary, had always enjoyed good health. (The patient had had a tracheotomy, and it was rather difficult for him to talk, and the son who accompanied him stammered; therefore it was under most adverse conditions that the following history was obtained.) Since coming to this country he has been working in the zinc mines of northern New Jersey. About 20 years ago he noticed a small lump on the right side of his neck. He paid little, if any, attention to this for it never gave him any discomfort or pain and did not incapacitate him in any way; notwithstanding, this lump had been gradually getting larger. About one and one-half years ago from date of history, March 16, 1940, the patient began to have some difficulty in breathing and swallowing and first consulted his family physician, who sent him to a specialist. On account of the patient's dyspnea, it was decided to do a tracheotomy as an emergency measure. This was done in February 1939, and relieved him somewhat of his dyspnea for a short time. As the growth developed, it interfered considerably with his swallowing, causing him to lose considerable weight. The swallowing became more difficult and his breathing more labored, despite the fact of having no tracheotomy tube.

On March 16, 1940, he was referred to me by Dr. Braun. Mirror laryngoscopy revealed a large smooth swelling in the right pyriform sinus, encroaching on the lumen of the trachea to such an extent that visibility of the cords was impossi-

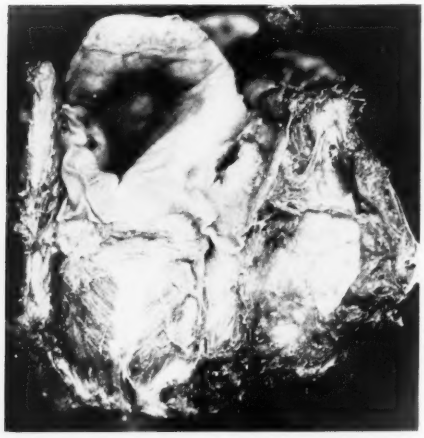

Fig. 3. Specimen showing larynx, ulceration, chondroma and hyoid bone, all removed en masse.

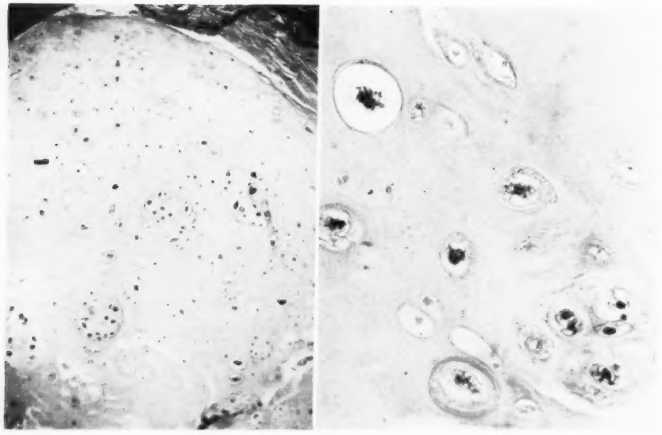

Fig. 4. A photomicrograph (X100) of chondroma of larynx. Note atypical nests of cells separated by hyaline intercellular substance.

Fig. 5. A photomicrograph (X400) of chondroma of larynx. Note atypical cells and irregularity of the nuclei.

ble, and also pushing the right lateral wall inward, interfering markedly with swallowing. On palpation no glands were felt, but there was a large smooth growth which was attached to the right thyroid ala, extending backwards almost to the cervical vertebrae.

The patient was admitted to the hospital, and a direct laryngoscopy was performed which revealed, on the right side, along the margin of the aryepiglottic fold, an ulceration, and pus was expressed therefrom; the larynx was completely closed by the swelling. From this ulcerated area, a specimen was removed for histological study, which showed necrosis with polymorphonuclear leucocytic infiltration and bacterial masses. The diagnosis was chronic ulcerative laryngitis.

In the absence of pain and gland involvement and with a negative histological examination for malignancy, a diagnosis of chondroma of the larynx was made, and preparations were made to do a laryngectomy with complete extirpation of the growth; this was done on April 3, 1940. At the time of operation, a large specimen was removed from the growth before the larynx was skeletonized. This report showed an area of cartilage cells, surrounded by fibrous tissue. The diagnosis was chondroma of the larynx. The operation was continued, removing the hyoid bone, larynx and growth en masse.

The patient's postoperative period was uneventful, and he was discharged from the hospital May 3, 1940. He has remained well ever since.

SUMMARY

A case of chondroma of the larynx is herein reported in which a total laryngectomy was necessary. Also citation of a case showing that chondroma of the larynx and carcinoma of the larynx may easily be confused. Chondroma is rare, and from the relatively few cases reported in literature it is obvious that not many laryngologists have had the opportunity to study these interesting cases.

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LI

TREATMENT OF CANCER OF THE LARYNX WITH EMPHASIS ON CONSERVATISM*

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In treating malignant tumors of the larynx complete eradication of the disease must always be the primary objective. Preservation of laryngeal function is of secondary importance but is essential if it will not jeopardize the likelihood of cure. Although the most extensive surgical procedures possible are frequently required in order to remove completely cancer in this situation, unnecessarily radical removal of laryngeal growths results in needless sacrifice of the voice and integrity of the respiratory tract. An open operation is the generally accepted procedure for removal of carcinoma of the larynx and is absolutely necessary in the majority of instances. However, in selected cases less radical external surgical procedures or even endoscopic measures will permit removal of the neoplasm without sacrifice of function and afford an equally good prognosis.

It is hoped that the title of this paper and the foregoing remarks will not convey the erroneous impression that function can be preserved in all cases of cancer of the larynx. Nothing could be more absurd nor further from the truth. As a matter of fact, in a large proportion of cases of cancer of the larynx encountered at the Mayo Clinic, laryngectomy is required for complete removal of the neoplasm. I do feel strongly, however, that laryngeal function should never be sacrificed unnecessarily in these cases. A patient should not be doomed to carry on respiration through a tracheal tube for the remainder of his life without the most careful study and serious consideration on the part of the surgeon. The generalizations encountered in the literature which state that carcinoma involving the anterior commissure, the false cord or ventricle should not be removed through a laryngofissure but necessitate laryngectomy are not entirely substantiated by facts. Actually, it is often feasible to remove completely such lesions from these situations by means of thyrotomy. The fact that laryngofissure performed by experienced

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laryngologists yields more than 80% of the five-year cures bears evidence to the effectiveness of such removal.

FACTORS INFLUENCING CHOICE OF TREATMENT

The common classification of malignant laryngeal tumors as intrinsic and extrinsic growths is of little value in determining the method of treatment advisable in these cases. In fact, only the broadest generalizations pertaining to methods of removal can be based on any classification of these neoplasms founded on their situation, for the procedure indicated in a given case is dependent on a number of additional factors. The extent of the lesion is of the utmost importance; at operation this may be found to be much greater than was obvious on clinical examination. Perforation of the thyroid cartilage or of the cricothyroid membrane may be present, and although such extension is usually obvious grossly, the inflammatory infiltration accompanying it may render it unrecognizable without histologic examination. In this connection, fresh frozen sections for microscopic study are of the greatest value. On the other hand, the lesion may be more limited in extent than was apparent on clinical examination, so that more conservative removal than was anticipated is possible.

The activity of the tumor must always be given serious consideration. This is emphasized by the work of New and Fletcher who demonstrated the marked discrepancy between the gross and microscopic limits of the disease in lesions of high grade. Previous treatment and acute inflammatory reaction in the lesion are also of importance, for both render wider removal of the lesion imperative even though microscopic examination proves the lesion to be of low grade or of only moderate activity. The age and general condition of the patient too are factors to be reckoned with; obviously, radical surgical procedures should not be undertaken if the patient's life expectancy is not sufficient to justify them. The necessity of biopsy in these cases cannot be overemphasized. Radical measures for removal of a neoplasm of the larynx should never be carried out without microscopic proof of the malignant nature of the growth. Every laryngologist of wide experience has been surprised on more than one occasion to find on histologic examination that a lesion which clinically had appeared to be frankly malignant was actually inflammatory. The possibility that removal of a specimen for biopsy when properly carried out produces any deleterious effect seems too remote to warrant consideration. Performance of the procedure in

more than 1,000 cases of carcinoma of the larynx at the Mayo Clinic has failed to reveal any evidence of this.

METHODS OF TREATMENT

The therapeutic measures of value in malignant tumors of the larynx include thyrotomy, laryngofissure, laryngectomy, pharyngotomy, endoscopy and irradiation. The first two of these are the procedures generally employed in the removal of such growths. Pharyngotomy, endoscopic measures and primary irradiation are indicated in only a comparatively small group of selected cases. The proportion of laryngofissures and laryngectomies in any given series of cases of cancer of the larynx varies greatly, depending on the stage of the disease and on the training and experience of the laryngologist. Prior to the past two decades laryngectomies comprised the great majority of these procedures in most clinics. The chief reasons for this were that (1) the early symptoms of laryngeal malignant lesions were not generally known and such lesions were not recognized until well advanced; (2) cases in which thyrotomy was performed were not properly selected and recurrence developed in a high percentage; (3) the technic of the operation was not well developed and the mortality rate was high; and (4) a number of eminent laryngologists felt that thyrotomy should be limited to cases of incipient laryngeal cancer only. MacKenty expressed well the pessimistic attitude held by many laryngologists in this connection when he stated that as experience accumulated, the sphere of thyrotomy contracted. In recent years this situation has changed considerably, and the converse of this is largely true at present. The proportion of thyrotomies to laryngectomies has increased so that in many series the less radical procedures far outnumber the latter. Of 135 operations for carcinoma of the larynx reported by New and Waugh in 1934, 75 were thyrotomies and 60 were laryngectomies. The preponderance of laryngofissures is only slightly less than this at present.

Both thyrotomy and laryngectomy recently have undergone changes in the hands of certain surgeons that have affected not only the scope of these operations but their effectiveness as well. Some laryngologists have rendered laryngectomy more conservative by stripping the outer perichondrium from the surface of the thyroid cartilage, thus making the procedure a subperichondrial resection. The aim of this change has been to lessen the severity of the postoperative reaction. It is definitely stated that the operation is not suitable when the cartilages are invaded, the false cord is involved or the growth has spread to the muscles and glands of the neck. In

our experience, involvement and even perforation of the thyroid cartilage or cricothyroid membrane frequently cannot be recognized preoperatively by clinical examination, roentgenographic studies or by any other means. In these cases the condition is found at operation, and opening into it is avoided only by the routine practice of carefully dissecting the muscles from the anterior and lateral surfaces of the larynx. In stripping the perichondrium from the cartilage in such cases the neoplasm is almost certain to be opened. Although this is not disastrous, if recognized immediately and thorough cauterization of the involved region performed, it should be avoided if possible. Experience in a number of such cases has convinced me of the feasibility of effectively removing the lesion when such histologic involvement is present. On numerous occasions, limited invasion of the muscles has been found while these structures were being dissected from the thyroid cartilage. The involved region has immediately been electrocoagulated thoroughly in order to produce an eschar to protect the wound from the lesion, and then wide electrosurgical excision of the adjacent tissues has been carried out along with removal of the larynx. Most of these patients have not had unduly severe postoperative reactions, and many of them have remained free from recurrence of the growth. When such involvement is suspected prior to operation, excision of the overlying soft tissues is made primarily; if necessary, all of the muscles and subcutaneous tissues anterior to the larynx are excised. It is only fair to state in this connection that statistics covering laryngectomy carried out in a group of cases, including a number of such extrinsic lesions, cannot be compared with those obtained in treating cases many of which might well have been taken care of with thyrotomy. However, a number of patients have been cured of advanced malignant disease by this means who probably would not have been cured otherwise. On the other hand, the scope of laryngofissure has been increased by rendering the procedure more and more radical. The operation in some cases amounts almost to hemilaryngectomy, except that the continuity of the cricoid cartilage is not disturbed.

Since it is frequently impossible to determine definitely by clinical examination the limits of the malignant process within the larynx, decision as to the surgical procedure necessary for its removal must be deferred until direct inspection of the growth is made through a laryngofissure. At the clinic we carry out exploration of this type in all cases in which the growth is not well defined, and, as previously noted, often it has been possible to remove through this approach lesions that at first appeared to require total laryngectomy.

A case in point that stands out strikingly is that of a man of advanced years who had a sarcoma which completely filled the larynx from the level of the tip of the epiglottis above to beyond the lower border of the cricoid cartilage below. On exploration through laryngofissure, the neoplasm was found to be attached by a comparatively small pedicle to the superior surface of one vocal cord and the adjacent floor of the ventricle. This attachment was thoroughly electrocoagulated following removal of the tumor. The patient has now been well for eight and a half years and possesses a normal voice.

If examination through a laryngofissure discloses that the lesion is too extensive to permit safe removal by this route, laryngeal forceps are applied to the margins of the divided thyroid cartilage, and laryngectomy is carried out. As the growth itself is not opened into and traumatized, there is no contamination of the wound other than that caused by laryngeal secretions, which cannot be avoided. It is possible to remove much more extensive growths through a laryngofissure than is generally believed. MacKenty performed laryngectomy for lesions which involved more than the surface of the middle third of one vocal cord or ventricular band. However, the idea that such radical removal is necessary has not been shared by most laryngologists. In fact, Thomson, who for many years has been one of the most ardent supporters of thyrotomy, pointed out that although the most favorable cases are those in which the disease is limited to one vocal cord, particularly the anterior portion, good results are obtainable even when the disease has spread across the anterior commissure so that all of one cord and a portion of the other have to be excised. My own observations are in keeping with this. On a number of occasions I have removed all of one vocal cord and the anterior half of the opposite cord through a laryngofissure. At times in such cases, approximately the anterior half of the thyroid cartilage on both sides has been excised without interfering seriously with function subsequently.

Sharp excision is rarely depended on alone in removal of cancer of the larynx through laryngofissure even when the growth is small. Electrocoagulation has proved to be an extremely valuable adjunct in this connection and is applied routinely to the laryngeal wound after excision of the lesion. This procedure not only effects wider removal of the neoplasm but guards against possible grafting of malignant cells. The eschar definitely delays and often appears to prevent invasion of the freshly opened tissues by the organisms present in the respiratory tract. The duration of the protective period varies considerably but is definitely prolonged by painting the coagu-

lated surface with a solution of acriflavine and gentian violet and then coating it lightly with petrolatum. As a result, less acute local reaction is likely to occur immediately after operation, and as the tissues are given an opportunity to build up a defensive granulation barrier, the late reaction often is less severe. Electrocoagulation of exposed cartilage has not increased the likelihood of perichondritis. However, most of the coagulated cartilage usually is removed either by sharp excision or by means of rongeurs in order to decrease the size of the sequestrum. It is occasionally necessary to remove sequestrated cartilage later. Aspiration of sequestrated cartilage in cases of this type has not been encountered, although this occurred in one case of perichondritis following irradiation of multiple papillomas of the larynx.

Implantation of radium points at times is made directly into the wound following removal of an extensive epithelioma through laryngofissure. Platinum points containing 1 mg. of radium element are usually employed; these are packed in the wound with iodoform gauze. Since the overlying cartilage has usually been excised and care is exercised to keep the radium at a safe distance from the remaining cartilaginous surfaces, no difficulty with perichondritis has occurred in these cases at the clinic. Only moderate dosage is usually employed, although intensive dosage could probably be given safely if indicated.

On one occasion at the insistence of the patient an extensive fixed carcinoma of moderate activity which involved one side of the larynx was removed through a laryngofissure when it was felt that laryngectomy should have been performed. The procedure was carried out under protest, for on direct inspection it did not appear possible to remove the growth completely with this approach and the patient refused to submit to laryngectomy. The overlying portion of the thyroid cartilage was removed following thorough electrocoagulation of the growth. The local postoperative reaction was protracted, but in a recent communication received more than five years after operation, the patient stated that his throat had given no further trouble and he was able to carry on his dental practice.

In some of these cases the arytenoid on the affected side together with so much of the thyroid cartilage has been excised that collapse of the larynx has been feared. This has not occurred in a single instance; the airway always has been ample and in the course of time a surprisingly good voice has developed. Removal of the arytenoid cartilage carries with it grave risk of aspiration pneumonia

because of loss of this sphincter action at the level of the glottis during the patient's convalescence. This complication has occurred in a few instances. It seems surprising that it does not occur more frequently, for infected secretions of the throat and drainage from the laryngeal wound tend to gravitate directly through the open glottis. An iodoform gauze pack firmly placed in the larynx and allowed to remain for a few days to a week or more, or until the tissues adjacent to the wound have recovered to a certain extent from the sensory and motor disturbance resulting from the operation, is of definite value in this regard. The small amount of secretion that seeps by the pack appears to be well taken care of by the cough reflex; little trouble has been experienced at this stage in most of the cases. Later when healing is complete, examination of the larynx reveals a definite funnel-effect similar to that which appears following complete removal of the epiglottis. Yet no difficulty has been encountered with deglutition, as has also been true in cases in which the epiglottis is removed. The satisfactory readjustment that nature is able to make in such cases has been a surprising observation. From the appearance of the throat one might well expect that the patient would be continuously annoyed by the aspiration of secretions, but no appreciable difficulty is experienced.

Many laryngologists feel that removal of malignant tumors from the larynx by other than an open operation is wholly unjustifiable, and, in general, this is a safe rule to follow. In 1920, Lynch reported before the American Laryngological Association a group of cases of cancer of the larynx in which he dissected out the lesions under suspension laryngoscopy, with good results. Unquestionably, many laryngologists on reading his report shared my feeling of doubt concerning the wisdom and effectiveness of the procedure. However, after cautiously using it and observing results during the course of a number of years, it can be endorsed heartily for use in a carefully selected group of cases. Many malignant neoplasms that are situated in the supraglottic portion of the larynx can be removed just as effectively, and often more safely, through the mouth than by an external operation. In most cases it is possible to visualize such lesions satisfactorily by means of direct, preferably suspension, laryngoscopy. Their removal with this approach by means of electrocoagulation is not only feasible but is simpler, safer and just as certain as removal through pharyngotomy.

Most of the supraglottic lesions originate on the epiglottis and secondarily involve the aryepiglottic folds; a few are primary in the latter situation and a few on the posterior or posterolateral aspect of

of the arytenoids. Those which develop on the epiglottis arise more commonly from the posterior aspect and are usually fungating rather than infiltrating. They tend to grow slowly and metastasize late, in contrast to many of the tumors which originate on the base of the tongue and involve the epiglottis secondarily. Because of the slight infiltration usually present about the base of these tumors, treatment is effective in eradicating the lesion in a high percentage of cases even when the growth is apparently well advanced. In some of the cases, the tumor filled most of the lower portion of the hypopharynx and completely obscured a view of the epiglottis on both direct and indirect inspection, yet it was entirely eradicated by electrocoagulation applied through the mouth. On the other hand, neoplasms which arise from the arytenoids and pyriform fossae are usually highly malignant and prone to metastasize early. Such lesions should be dealt with in this manner only if they are seen early and are well localized. Postcricoid lesions usually cannot be visualized well enough to permit removal through the mouth.

A small group of patients are not satisfactory subjects for procedures which necessitate any type of direct laryngoscopy, for even though complete relaxation is secured by means of a general anesthetic, the anatomic structure of the throat will not permit visualization of the anterior portion of the larynx.

Suspension laryngoscopy as carried out at present with cocainization of the throat and intravenous anesthesia with pentothal sodium is much easier and safer than it was with the deep ether anesthesia formerly employed. In an occasional case, however, ether must be administered to supplement the intravenous anesthesia in order to secure complete relaxation.

If the lesion under consideration is extensive, preliminary tracheotomy is advisable because of the possibility that sufficient edema may develop postoperatively to produce respiratory obstruction. The local reaction following tracheotomy is permitted to subside before treatment of the tumor is undertaken.

The risk associated with removal of the tumor under suspension laryngoscopy appears to be slight. There has been little immediate postoperative reaction in cases in which this procedure has been used, and convalescence has been surprisingly mild and brief. There is practically no trauma to the tissues aside from that caused directly by the electrocoagulating process; this is, of course, to be expected. Approximately two and a half to three weeks are required for separation of the slough unless implantation of radium points is used to

supplement electrocoagulation; the local reaction is then much more protracted. Bronchopneumonia, apparently attributable to aspiration of slough, occurred in one instance but was followed by recovery. To date, secondary hemorrhage has not been encountered in any of the cases.

Functional results have been satisfactory in all cases, and there has not been any impairment of the voice nor difficulty in swallowing as might be anticipated following complete removal of the epiglottis together with a portion of the aryepiglottic folds.

In some instances when the malignant tumors in the supraglottic portion of the larynx are infiltrating and extend beyond the epiglottis and especially when satisfactory visualization by means of laryngeal suspension is not possible, an open operation, either pharyngotomy or thyrotomy, must be carried out. Laryngofissure affords excellent exposure of tumors that are situated laterally or posteriorly above or below the glottis but confined within the larynx. However, pharyngotomy usually furnishes the best view of the supraglottic tumors, especially of those located anteriorly. The choice between subhyoid and transthyroid pharyngotomy depends on the situation and extent of the lesion. Tumors which involve the epiglottis and extend posteriorly along the aryepiglottic folds and laterally into the pyriform fossae are visualized better through a subhyoid approach; this is done by incising transversely at the base of the tongue and drawing the epiglottis out into the wound. When there is limited involvement of one aryepiglottic fold with extension onto its outer aspect or when the epiglottis and base of the tongue are involved, lateral pharyngotomy after removal of the thyroid ala and, if necessary, a portion of the hyoid bone is preferable if not absolutely essential. The pharynx usually is opened on the side where the lesion is located, but in some instances an opening on the opposite side permits more satisfactory exposure. The lesion usually is removed by means of electrocoagulation, and if deemed advisable this procedure is supplemented with interstitial irradiation. Preliminary tracheotomy may or may not be necessary. As these lesions do not as a rule involve the vocal cords, interference with laryngeal function usually does not occur following healing.

Prior to a few years ago, indirect laryngoscopy was used almost entirely at the Mayo Clinic among adults for removal of localized benign laryngeal tumors and for other minor intralaryngeal procedures. If the patient is co-operative and the larynx well cocainized, it is possible for a surgeon accustomed to this type of work to perform rather extensive procedures satisfactorily in this manner.

REPORT OF A CASE

In March, 1927, a man 43 years of age came for consultation after he had been advised that laryngectomy was necessary as biopsy of a lesion on his vocal cord had revealed squamous cell carcinoma. Examination of the larynx at the clinic revealed a region of recent trauma on the middle portion of the right vocal cord but no evidence of neoplasm. The tissue previously removed was obtained, and microscopic examination confirmed the previous diagnosis of squamous cell carcinoma Grade II, on a grading basis of I to IV. Observation several weeks later after subsidence of the inflammatory reaction revealed a lesion that clinically appeared to be an epithelioma situated on the superior surface of the middle third of the right vocal cord which measured approximately 4 mm. in diameter. This lesion did not involve the free border of the vocal cord. Because of its limited extent and low grade of malignancy the lesion was electrocoagulated in situ. Indirect laryngoscopy and local anesthesia were used. Little local reaction developed following this procedure. Recent examination, more than thirteen and a half years after removal, did not reveal recurrence of the growth. The patient's voice was about normal, although its range was narrower than previously.

This case is cited merely to show by what a simple procedure a malignant lesion of the larynx can be eradicated. However, it must be stressed that rarely is it feasible or justifiable to attempt removal in this manner.

Surgical removal and irradiation, the two accepted methods of treatment of cancer of the larynx, can be used either alone or combined. Each has its definite indications, but as Martin has pointed out, they frequently are emphasized only from their competitive standpoint. Radiologists and laryngologists generally agree that carcinoma which is limited to the interior of the larynx should be dealt with surgically, and frequently it is advantageous to supplement this with irradiation. Also, irradiation is the treatment of choice when the lesion involves the regional lymph nodes or extensively invades the surrounding muscle and when the growth arises in the postcricoid region or the pyriform fossa.

By far the most impressive results from the irradiation of cancer of the larynx have been obtained with fractional dosage roentgen therapy applied according to the method of Coutard or a modification of this. In some instances in which this method of treatment has been used for patients who had inoperable lesions or who, because of their advanced age or poor general condition, could not be subjected to radical surgical procedures the immediate results have been almost miraculous. At times when an extensive lesion had been producing such marked respiratory obstruction that tracheotomy was necessary before irradiation could be given, the growth has entirely disappeared after irradiation; the larynx has returned to normal ap-

pearance and function, and the tracheal opening could be allowed to close. In other cases in which it appeared that the carcinoma would be fatal within a few months, the disease has cleared completely after irradiation, and the patient has been restored to apparently normal health. In some of these cases the growth has not recurred during the two or three years that the patients have been observed. More frequently the improvement and apparent cure described have been of only a few months' duration, and the neoplasm has then progressed to terminate fatally in spite of repetition of treatment.

The immediate reaction following this treatment is often severe, prolonged and painful. Extensive denuding of the larynx and hypopharynx as well as of the skin overlying these structures is usual. Among patients who have thin skin and those who possess little cutaneous pigmentation, the reaction from irradiation is likely to be exceptionally severe. Sloughing within the larynx possibly with secondary hemorrhage and aspiration pneumonia also may occur.

Sufficient time has not yet elapsed to permit satisfactory evaluation of this form of treatment and comparison of its efficacy with results of surgical measures in such cases, nor can the late effects of treatment as regards delayed perichondritis and possible development of chronic actinodermatitis be anticipated. Suffice it to say that although the primary results from irradiation are at times striking, they are still too uncertain and inconsistent to justify the use of this form of treatment alone when the lesions can be removed surgically.

CONCLUSIONS

1. Unnecessarily radical removal of malignant tumors of the larynx at times results in needless sacrifice of the voice.

2. The method of treatment advisable in a case of carcinoma of the larynx often cannot be determined until the growth is inspected directly through a laryngofissure.

3. The more conservative type of laryngectomy now performed by some laryngologists really deprives patients with involvement or early perforation of the thyroid cartilage of the benefit of the operation.

4. Thyrotomy is effective in treatment of more extensive laryngeal malignant growths than is generally believed.

5. Electrocoagulation is a valuable adjunct in the removal of carcinoma of the larynx through laryngofissure, pharyngotomy or suspension laryngoscopy.

6. Lesions which involve the supraglottic portion of the larynx can often be removed by means of suspension laryngoscopy and electrocoagulation, supplemented with insertion of radium if necessary.

7. Extensive supraglottic lesions that cannot be visualized in their entirety by suspension laryngoscopy require pharyngotomy.

8. Fractional dosage roentgen therapy is the most effective form of irradiation in the treatment of cancer of the larynx, but the results are still too uncertain to justify its use in place of surgical procedures when surgical removal is possible.

MAYO CLINIC.

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LII

LARYNGEAL TUBERCULOSIS WITH SOME OF THE MORE RECENT ADVANCES IN TREATMENT

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BOULDER

This article has been written at the request of the editors. This is an attempt to bring the subject of treatment up to date. In spite of the occasional thoughtless statements that there is nothing new in the diagnosis and treatment of this disease there are many advancements. While these are not as far reaching and miraculous as the treatment of diphtheria with antitoxin, the control of diabetes with insulin and the treatment of streptococcic infections with sulfanilamide these are none the less definite advancements.

This is not at attempt to record the history of tuberculosis beginning with the time of Hippocrates nor is it an attempt to quote all the articles which have been written to date in all languages. Such a paper would be little more than a catalogue, as there are now approximately 3,000 references in 27 languages to this one disease.

Laryngeal tuberculosis always has been and is now in almost every instance secondary to pulmonary tuberculosis. Most of the case reports of primary tuberculosis of the larynx have been so reported because the physician diagnosed the laryngeal complication before the clinical signs and symptoms of the pulmonary disease became manifest. With modern methods of diagnosis supplemented by the laboratory and the roentgen ray there is less likely to be such a report. While it is conceivable that there may be primary cases these must be extremely rare. Academic discussion has its place, but for the busy laryngologist factual presentation is much more important.

ETIOLOGY

The role of the tubercle bacillus is accepted without question, but in recent years the infection of the larynx by metastasis from the lungs has more advocates than formerly. Whether the larynx became infected by contact with sputum-laden bacilli or by metastasis is very difficult to prove even at autopsy. The investigations

carried out by Spencer and Summerill[1] with autopsy specimens showed that nearly all the structures and organs of the upper respiratory tract contain a few tubercle bacilli even in patients who showed no clinical gross evidence of tuberculosis of this region before death.

PATHOLOGY

The clinical lesions are naturally of most importance to the laryngologist who has to make the diagnosis, differential diagnosis and carry out the many minute details of treatment in conjunction with the internist and even the chest surgeon. The following classification is practical from the clinical point of view:

1. Vascular changes.
   a. Anemia.
   b. Hyperemia.
2. Infiltration.
3. Tuberculoma.
4. Ulceration.
   a. Acute or active.
   b. Chronic or sluggish.
5. Edema.
6. Fibrosis.
7. Perichondritis.
8. Chondritis.
9. Necrosis.

The usual classification of acute, subacute and chronic must also be taken into account. Laryngeal tuberculosis is almost always chronic from the beginning, but acute and subacute exacerbations are by no means uncommon. In practice we all see occasional cases which do not conform to any classification, but this is true of most diseases and should be anticipated.

Dworetzky[2] advocated the following classification:

Peracute tuberculosis of the larynx.

Acute tuberculosis of the larynx { Stage 1. Stage 2. Stage 3.

Subacute tuberculosis of the larynx { Stage 1. Stage 2. Stage 3.

Chronic tuberculosis of the larynx { Stage 1. Stage 2. Stage 3.

Fetterolf[3] classified the ulcerative stages as follows: infiltration with superficial ulceration; infiltration with deep ulceration; infiltration with superficial and deep ulceration.

Histopathology.—The tubercle with giant cells and caseation necrosis suggests a tuberculous infection. If acid-fast bacilli, epithelioid cells and lymphocytes are found with an absence of plasma cells the diagnosis is complete. Giant cells may be the result of irritation and may be found in nontuberculous lesions.

SYMPTOMATOLOGY

The first subjective symptom may be slight hoarseness. This may be present only in the morning or after a paroxysm of coughing. Talking may be followed by hoarseness. The patient may say the throat feels full or it may burn and smart. Pain is not likely to be an early symptom. Fruit acids, tomato juice, vinegar, etc. may cause some discomfort or even choking. The throat may have to be cleared oftener and there may be more cough. Fatigue is more likely to occur early after talking for only a short time. Patients may be conscious of a greater effort required for talking.

Hoarseness may be followed in a short time by aphonia and there is greater difficulty in swallowing. After some weeks or even months swallowing may become painful. Severe pain is almost always a late symptom, and any pain is very unfavorable. It seriously interferes with proper nutrition which is so important if the patient is ultimately to recover. Pain usually means a poorer prognosis.

A few patients may be found with even a moderately advanced laryngeal tuberculosis without any subjective symptoms. However, this is the exception to the rule. In any event it shows the necessity for early and repeated laryngeal examinations as a preventive measure.

Dyspnea is often complained of if there is any narrowing of the lumen of the larynx. It can become a very serious symptom if there is a bilateral abductor paralysis.

The objective symptoms are not always striking at first. There may be a slight anemia, because most tuberculous patients have secondary anemia. The larynx on the other hand may be red, especially if the patient is coughing a great deal. Infiltration at the interarytenoid sulcus is an evidence of early tuberculosis. The anemia and redness are not tuberculous. If the infiltration extends

to the arytenoids these become larger and may be club-shaped. During phonation it is easier to see the infiltration. A small, irregular pale mass may project from the infiltrated posterior laryngeal wall. This resembles a papilloma, but it is a tuberculoma.

Ulcers may form as the infection of the larynx becomes more severe or more extensive. These are likely to be superficial at first and later deep. If a tubercle ruptures from caseation the ulcer is deep.

Edema is a late sign and means the circulation is interfered with, although it is not a true edema. Sir St. Clair Thomson[1] calls this pseudo-edema. It produces an ashen-gray appearance. This has often been called pathognomonic of laryngeal tuberculosis.

Fibrosis is nature's attempt to heal the tuberculous process. It is the same healing process which takes place in the lungs. Without it the patient goes from bad to worse. If there is too much fibrosis there may be cicatricial stenosis.

Perichondritis and chrondritis are usually rapidly fatal. These are very late symptoms and are very painful. Swallowing is next to impossible when the laryngeal cartilages become involved.

DIAGNOSIS

The finding in the larynx of any of the preceding lesions should at least make one consider laryngeal tuberculosis. The diagnosis cannot be certain, however, without a general physical examination, roentgen ray examination of the chest, blood and sputum examinations. The urinalysis is, and should be, routine for all patients.

Differential Diagnosis.—The blood or spinal fluid Wassermann, the Kahn or the Hinton test are indicated. A history of syphilis is not often easy to obtain. Luetic lesions in other organs help to clinch the diagnosis. If in doubt about malignancy a biopsy is often necessary. Tuberculosis of the larynx is most frequently found in adults from 20 to 45 years of age. It may occur earlier or later. It is remarkable how few children and relative few adolescents have laryngeal tuberculosis. Syphilis is more likely from 18 to 40 and carcinoma after 50 years of age. The latter is more frequent in men. The lungs and sputum are usually negative with lues, although any two or all three of these diseases have been present in the same patient and at the same time. Syphilis and carcinoma have a predilection for the anterior half of the larynx while tuberculosis, at least early, is found more frequently in the posterior half.

However, there are exceptions to this, and rules are not to be depended upon too much. The most thorough examination possible is to be preferred.

PROGNOSIS

The outlook for the patient necessarily depends upon the amount of laryngeal involvement, the duration of the involvement, the extent of the pulmonary tuberculosis, the duration of the pulmonary disease and last, but not least, the ability of the patient to gain in spite of the laryngeal complication. The laryngeal disease is very rarely an early complication of the pulmonary tuberculosis, and tuberculosis in all its forms and in all organis is usually chronic. In other words the pulmonary disease had likely been present several years before the larynx became infected. Laryngeal tuberculosis is likely to make ultimate arrest of the disease or recovery more difficult and tedious. It immediately makes the prognosis less favorable. Spencer[5] in 1924 and Greene[6] in 1937 have shown how many conditions can and do influence the prognosis. These all require due consideration from time to time as the patient improves or gradually fails. The prognosis changes as the patient's condition changes for better or worse.

TREATMENT

So many remedies have been recommended and used for the treatment of the larynx that this shows we do not have a specific remedy. It is not necessary to even mention in this article all of these. Only those procedures which have proved to be of merit or which give promise of merit will be discussed.

Cauterization heads the list as the most important procedure. Wood[7] in 1911 strongly urged the actual cautery, and he deserves great credit for making it a standard procedure even though it had been recommended and used before. This may be used by the direct or indirect method, but the latter is usually the better and under local anesthesia. Sir St. Clair Thomson[1] emphasized the importance of using the cautery point of platinum at white heat in order to avoid pulling the escharotic tissue out with the cautery. This avoids leaving a bleeding area instead of a sealed area at the point of cauterization. Several lesions may be cauterized at one time if the patient tolerates the cauterizations well. Patients with high fever and very active pulmonary tuberculosis are usually not suitable for the use of the cautery.

Vocal Rest.—Rest for the infected larynx is just as important as rest for the infected lung. Whispering is better than talking, but absolute silence is the only certain way to put the larynx at rest. Patients must use a pad and pencil to communicate with laryngologists, internists and nurses. For some patients this is rather depressing, but for those who are willing to make the sacrifice the larynx usually improves.

Heliotherapy.—The chemical, actinic or ultraviolet rays of the sun are the tanning rays and do much to improve the general health of the tuberculous. In cities or climates where the sun's rays are not available the ultraviolet lamp will have to be used. When sunlight is available the Verba laryngoscope will reflect the actinic rays into the larynx. The mirrors are made of an alloy of aluminum and magnesium so that they will not absorb the chemical rays. The patient must be out of doors, because glass or even dusty screen will absorb the ultraviolet rays. The larynx should be exposed to the rays for one-half to one minute daily the first week and the time gradually lengthened to not to exceed 10 minutes for most patients. Most people believe if a little is good more will be better and they use too much. The larynx is more easily sunburned than the skin. The tolerance and the maximum dose vary greatly with different patients. The laryngologist must direct the treatment. He can do this only by studying the patient and watching the larynx. Rules are, after all, only relative.

Oil Sprays and Drops.—Mineral and animal oils have been recommended from time to time. These are soothing to an irritated larynx, but are almost entirely palliative. Chaulmoogra oil in 20% solution may be dropped into the larynx with a Lukens syringe. Lukens,[8] LaRue[9] and others have reported their results from the use of this oil.

A solution of ½ to 2% menthol in liquid petrolatum may be used in the same way. Too much must not be used. The danger of lipoid pneumonia must be considered.

Injection of Alcohol.—This is used for the relief of pain and is not curative. This is carried out as follows: the skin of the neck should be washed with alcohol over an area covering the larynx and adjoining cervical region. Tincture of iodine or tincture of merthiolate may be painted on the skin at the site selected for the injection in place of the alcohol. Three cc. of a 50% aqueous solution of ethyl alcohol should be drawn into a sterile syringe, as 2 or 3 cc. should be injected on each side. The patient should be placed on a

bed or table with the head extended, so as to put the cervical muscles on the stretch. The superior laryngeal nerve can usually be located at about 3 cm. from the thyroid notch, on each side, as it passes over the upper border of the thyroid cartilage to enter the larynx. Pressure at this point, with the finger nail, usually elicits pain. The patient should be told he will suffer a little pain, but that it will not be severe and will not last long.

The needle should be slowly plunged through the skin at a right angle to the surface for a depth of 1½ cm. and the needle point moved around until the patient complains of sharp pain in the ear, jaw, neck or arm on that side, though an occasional patient will not complain of any pain. The solution should be injected slowly while the needle point is being moved about. After withdrawing the needle the puncture should be sealed with flexible collodion. The injection may be repeated on the other side at once or at some subsequent time according to the patient's condition and the laryngologist's surgical judgment. The injection of alcohol has been recommended by Hoffman,[10] Sturmann,[11] Fetterolf,[12] Vanderhoof,[13] and others.

Ionization.—Grain[14] in 1936 and Cotton-Cornwall[15] in 1938 recommended ionization for the relief of pain in tuberculous laryngitis. Grain encircles the larynx with special electrodes, employing a galvanic current of 10-12 ma. for 30-40 minutes. The positive pole is wet with water and placed on the back of the neck. The anterior or negative pole is placed over the larynx and is wet with a 2% potassium iodide solution. The electrodes are protected by layers of absorbent cotton extending beyond the edges. These treatments are given daily until the anesthesia lasts 24 hours. After this they are repeated only when the pain returns. There is a feeling of constriction in the neck according to Grain and a taste of copper on the tongue. The former lasts only four or five minutes, and the latter throughout the treatment.

The anesthesia begins early and is total according to Grain. It is progressive and lasts longer after each treatment. He believes it is not influenced by the size of the lesion and is stable. It has no value as a cure, as it only relieves the pain. The tactile sensation in the larynx is not disturbed, so that food does not enter the larynx.

Cotton-Cornwall's method of using ionization is so much the same as Grain's, at least in principle, that it will not be described.

Chemotherapy.—Sulfapyridine was used in 1939 experimentally for the treatment of guinea pigs infected with tuberculosis by Feld-

man and Hinshaw.[16] They began the administration of the drug before the animals were inoculated. They found the drug exerted a definite and striking modification and retardation of the expected course of the experimental tuberculosis in guinea pigs. At the time of their report they had examined the gross lesions but not the microscopic.

Spencer, Whitehead and Duffner[17] in 1940 published their results with sulfapyridine. They waited until the tuberculous lesions had developed in the guinea pigs before giving the drug. They obtained some improvement in the nodules and ulcers of the treated animals as compared with the untreated controls. The results were not as striking as those obtained in the treatment of streptococcic infections with sulfanilamide.

The greatest difficulty all investigators have in treating tuberculosis, in any form and in any organ, is to find a drug or chemical which will penetrate the tubercle bacillus with its 40% fat.

Tracheotomy.—This is sometimes a very necessary operation for patients with advanced laryngeal tuberculosis in order to keep them from choking to death. It prolongs life, but is not likely to effect a cure. The technic of this oldest of all operations need not be repeated here. It can be found in all standard textbooks on nose and throat diseases. Myerson[18] in 1935 reported his results in 708 cases of laryngeal tuberculosis over a three-year period. His article presents this subject exceptionally well.

Collapse Therapy.—The internist and the chest surgeon in recent years have greatly improved the treatment of pulmonary tuberculosis and have often prevented laryngeal tuberculosis. The question of when to use artificial pneumothorax, section of the phrenic nerve, section of the sympathetics, or thoracoplasty can best be decided by the internist and chest surgeon. However, laryngologists should encourage these methods of treatment for the prevention or arrest of the laryngeal disease. Probably no other method of treatment for pulmonary tuberculosis has ever done so much to prevent the laryngeal complication. Dr. Casper F. Hegner of Denver, a general surgeon and former head of the department of general surgery at the University of Colorado School of Medicine and Hospitals, has studied and developed chest surgery. He has done excellent work for the relief of patients and has saved many lives. Dworetzky[19] has done a great deal to show laryngologists the importance of collapse therapy.

Amputation of the Epiglottis.—If the tuberculous lesions are confined to the epiglottis and have not extended to the pharynx or base of the tongue, amputation of the epiglottis may arrest the disease. Late amputation only leads to disappointment. The operation may be performed by direct or indirect laryngoscopy and under local anesthesia. The latter method is preferable. The Moritz-Schmidt forceps, or some similar instrument, permit removal of the most of the epiglottis with one closure of the forceps. Reintroduction of the instrument is sometimes required.

Gastrostomy.—This operation may be required to keep patients with advanced laryngeal tuberculosis from slow starvation on account of painful swallowing. It helps to put the larynx at rest and lessens the odynphagia. It has been advocated by Lowen,[20] Cooper[21] and others. It prolongs life, but it is not likely to save the life of a patient with advanced pulmonary and laryngeal tuberculosis.

Numerous remedies and drugs could be listed for the treatment of this disease almost by the hundreds. Most of them have too little value to justify listing them.

PREVENTION

If the public would permit the use of one-half the scientific knowledge now at the physician's disposal, tuberculosis could be eliminated from the U. S. A. in another five years. The death rate has been reduced 64% since 1900. Too many patients are still unwilling to accept an early diagnosis. They, by their very indifference, insist upon waiting until they know they have the disease. This means more advanced lesions with the greater difficulty of obtaining an arrest or a cure. In the meantime they have infected other members of their family while they refuse to accept the facts. If it were not for the excellent sanitoria for the treatment of tuberculosis in nearly every state of the union this tragedy would be greater than it is. There are, on the other hand, many intelligent patients, who appreciate an early diagnosis, and welcome the prompt and early recovery which an early diagnosis usually assures.

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LIII

LARYNGEAL TUBERCULOSIS: A STUDY OF 500 CASES OF PULMONARY TUBERCULOSIS WITH A RÉSUMÉ BASED ON TWENTY-EIGHT YEARS OF EXPERIENCE*

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AND

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NEW YORK

By way of introducing the subject, I wish to state that the incidence of laryngeal tuberculosis is becoming smaller from year to year. During the past 28 years, I have made three detailed studies at the New York Municipal Sanatorium, Otisville, New York, one in 1914, another in 1934, and the present one in collaboration with Dr. Otto C. Risch in 1941. Groups of 500 patients, similar in the type of disease, were examined in each study, and the results as to incidence are as follows:

INCIDENCE RATE

TABLE I

YEAR	PULMONARY TUBERCULOSIS	LARYNGEAL TUBERCULOSIS	PER CENT
1914	500 cases	128 cases	25.6%
1934	500 cases	73 cases	14.6%
1941	500 cases	18 cases	3.6%

This reduction in the incidence rate of laryngeal tuberculosis can largely be explained by: 1) The ever-increasing educational cam-

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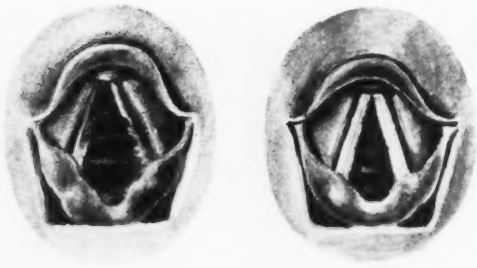


1. 2.

Fig. 1, Case 1. 1—H. W. Larynx before treatment. 2—H. W. Larynx after successful bilateral pneumothorax with Jacobeus on the right.

paigns leading to a greater number of case findings; 2) improvement in methods of examination by X-ray and laboratory technique, leading to earlier diagnosis and earlier treatment of pulmonary tuberculosis, for by thus controlling the pulmonary lesion, we are able to prevent the spread of the disease to the larynx; 3) improvement in the treatment of pulmonary tuberculosis and especially through the increasing employment of collapse therapy with the resultant beneficial effect on the larynx.

EDUCATIONAL CAMPAIGNS LEADING TO EARLY DIAGNOSIS AND TREATMENT

The conception of tuberculosis as that of a deadly disease, largely incurable or at best crippling the patient for life, has been entirely changed during the past two or three decades. This has been brought about in many ways. The numerous health campaigns waged by the National Tuberculosis Association and the talks given on the radio by men well qualified to speak on the subject, have educated the public to the importance of the early recognition of tuberculosis. The examination and X-raying of thousands upon thousands of contact cases at the health department clinics and at the offices of private physicians have rounded up many cases which might otherwise have escaped detection. The examination and X-ray of high school and college students have detected the disease in the adolescent before too great damage has been done. A compulsory examination and X-ray of nurses about to take up training have been required. Life insur-

ance companies, through a widespread advertising campaign, have made the man in the street health-conscious and have brought home to him in a forceful manner the economic value of good health. Business corporations have established a routine of annual or semi-annual health examinations of their employees, and in taking out hospital insurance, many applicants have been apprised of a hitherto unknown chest lesion. During recent months, the examination and X-ray of draftees for selective service has brought out many cases of pulmonary tuberculosis.

So through these various channels, the patient has learned that he has tuberculosis. In many instances, the disease is discovered in a verly early stage, and through this early diagnosis, half the battle has been won. The newer therapeutic methods of healing have now a better chance to bring about an arrest or a cure of the disease. Brilliant results have been achieved by three factors working in harmony: early diagnosis, careful management of the case in the hands of a properly trained physician of good judgment, and a cooperative patient. But the greatest of these is early diagnosis.

There is, therefore, no doubt that through the employment of educational campaigns and the improvement in diagnostic technique by the use of X-ray and improved laboratory methods, pulmonary tuberculosis is being discovered much earlier, and having been discovered, is being treated more efficiently and with greater success. The lesion often becomes checked before bacilli appear in the sputum, and laryngeal tuberculosis is thus prevented.

IMPROVED THERAPY

Another reason for the continued lessened incidence of laryngeal tuberculosis is that the treatment of pulmonary tuberculosis has become more aggressive as the years have gone by. Whereas in the past we relied to a great extent on bed rest and the improvement of the patient's resistance through hygienic and dietetic measures, we now, where the case permits, use collapse therapy. As soon as a diagnosis of pulmonary tuberculosis is made, our first thought is what more can be done for the patient in addition to bed rest. Today we are employing collapse therapy at the Otisville Sanatorium in 37.6% of the cases, and in my own practice in about 50%. In some institutions, it is employed in as high as 70-80% of all cases of pulmonary tuberculosis. In other words, as soon as a positive sputum is discovered, with or without cavity formation, we immediately think of collapse therapy. In this way, we have been greatly successful in

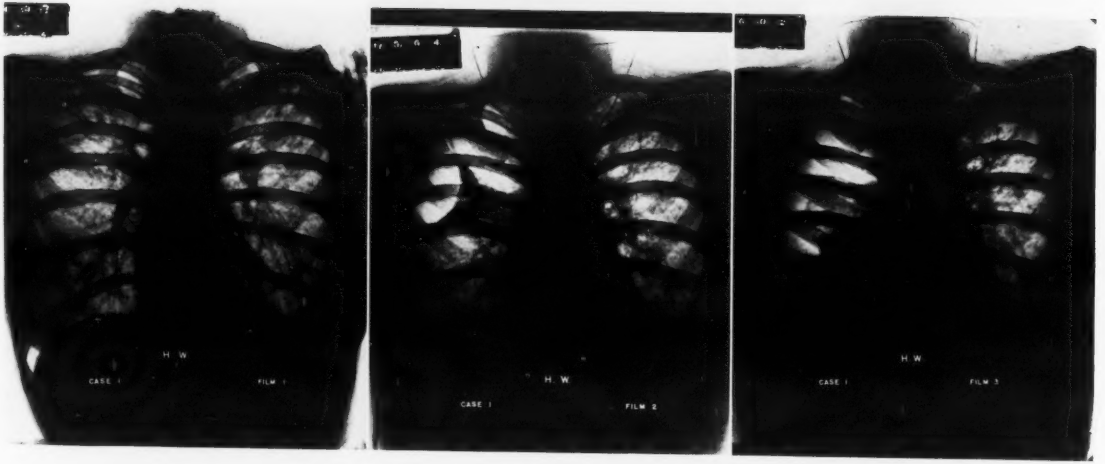


Fig. 2, Case 1.

Film I—Bilateral pulmonary tuberculosis with cavitations in right upper. Film II—Right sided pneumothorax with apical adhesion. Film III—After Jacobeus operation, closure of cavity.

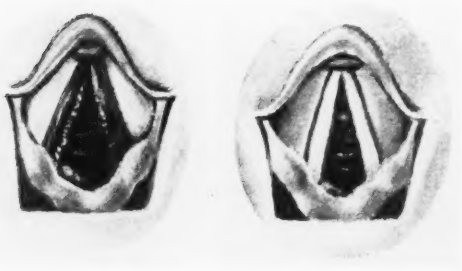
converting positive into negative cases. The above measures, of course, help to bring about an arrest of the pulmonary lesion, thus not only preventing, but also curing, laryngeal tuberculosis and thereby lessening the incidence rate.

In 1914 when I presented my first paper on laryngeal tuberculosis[1] before the New York Academy of Medicine, I examined 500 cases of adult pulmonary tuberculosis and found 128 of these, or 25.6%, had laryngeal involvement.

Twenty years later in 1934 I made a similar study[2] and presented my findings before the American Laryngological, Rhinological and Otological Society at Charleston, North Carolina. In that study of a group of similar cases at the Municipal Sanatorium at Otisville, I found that out of 500 cases, 73, or 14.6%, had laryngeal tuberculosis. I stated at that time that this pronounced reduction in the frequency of occurrence of laryngeal tuberculosis could readily be explained by improved methods of diagnosis and the consequent earlier application of treatment, and also by the newer methods of treatment. Whereas collapse therapy was not employed in any of the cases in 1914, it was employed in 20% of the cases in the 1934 group.

In the present study, a similar group of 500 cases was examined of which 440 were at the Otisville Sanatorium and 60 in my private practice. In this group of 440 cases at the Otisville Sanatorium, 7, or 2%, were found to have laryngeal tuberculosis. Of the 60 in my own private practice, 11 patients, or 18.3%, had tuberculosis of the larynx. The discrepancy in the incidence of occurrence between the Otisville group and the group in my private practice can easily be explained by the much larger number of advanced cases among the patients in my private practice (see Tables II and III), and also by the fact that several of these patients were referred to me because of laryngeal involvement. The total number of laryngeal cases among the 500 was 18, or 3.6%, which is a still greater reduction. This continued drastic reduction can again be explained by further improvement in our diagnostic methods and also by increasingly improved methods of treatment.

In summarizing, therefore, we may definitely state that improved methods of diagnosis and the increased application of collapse therapy are to a large degree responsible for the great reduction in the incidence of laryngeal tuberculosis as shown in Table I.



1. 2.

Fig. 3, Case 2. 1—F. S. Larynx before treatment. 2—F. S. Larynx after a Monaldi operation on the right side.

PATHOGENESIS OF PULMONARY TUBERCULOSIS

Since pulmonary tuberculosis always forms the background of laryngeal tuberculosis, I feel that it is pertinent to discuss briefly the pathogenesis of pulmonary tuberculosis and its relation to the various types of laryngeal lesions. For all practical purposes, we may classify all cases of pulmonary tuberculosis into two main groups: 1) the bronchogenic or broncho-pulmonary, 2) the hematogenous.

1. In the bronchogenic or broncho-pulmonary form the lungs are invaded by the tubercle bacillus through the air passages, and the pulmonary lesion spreads to other parts of the lungs, mainly by way of the bronchial route. This form comprises about 80% of all cases of pulmonary tuberculosis, and embraces all types of chronic tuberculosis, including exudative, caseous, fibrocavernous and productive.

2. In the hematogenous form, the tubercle bacilli spread through the lungs and other organs of the body by way of the blood stream. Such invasion of the pulmonary tissues probably occurs in the remaining 20% of all cases. Here it may be said that hematogenous tuberculosis may be very mild or severe, depending on many factors. Rubin,[3] in an excellent article on the subject, aptly states that "When tubercle bacilli invade the blood stream, there may result a generalized miliary tuberculosis that overwhelms the patient within a few weeks or there may be complete absence of symptoms. Between these extremes, there is a wide range for various manifestations of hematogenous tuberculosis in parts of organs, whole organs or groups of organs depending on the numbers and virulence of the

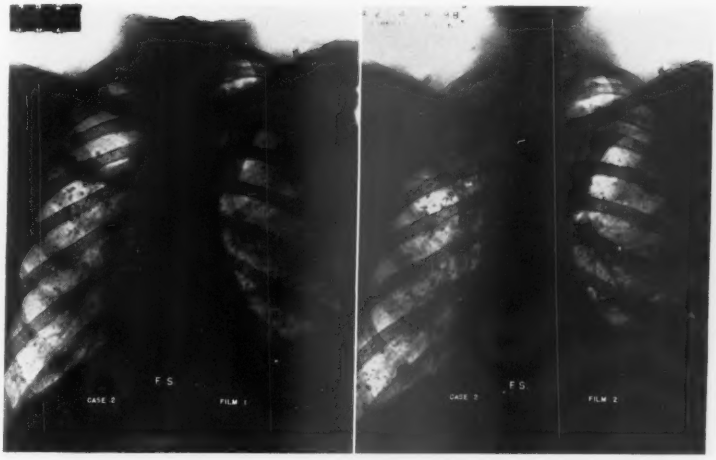


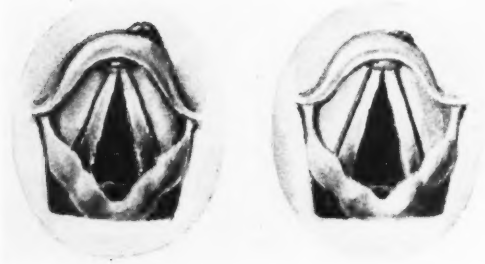
Fig. 4, Case 2.

Film I—Bilateral pulmonary tuberculosis with 10 cm. giant cavity in the right upper lobe. Film II—One year following Monaldi, cavity contracted about 80%.

tubercle bacilli, the frequency with which they invade the blood stream, the site where they enter the blood stream and the sensitivity of the body to tuberculotoxins."

Zavod[1] differentiates hematogenous tuberculosis from generalized miliary tuberculosis and states: "Pulmonary hematogenous tuberculosis is a pulmonary dissemination of the bacillus of tuberculosis by way of the lesser blood circle mainly, in contradistinction to generalized miliary tuberculosis which is a widespread dissemination of the bacillus of tuberculosis by way of both the greater and lesser blood circles."

Here it may be appropriate to note that although pulmonary tuberculosis may exist as bronchogenic or hematogenous during its early manifestations, after the pathological process is well-established, it often becomes mixed. A hematogenous lesion may ulcerate through the alveoli and become bronchogenic, while a bronchogenic lesion may break into the circulation and become hematogenous. In speaking, therefore, of a hematogenous or bronchogenic form, it is generally implied that the lesion is predominately hematogenous or



1. 2.

Fig. 5, Case 3. 1—M. F. Larynx before treatment. 2—M. F. Larynx following Monaldi operation and two stage thoracoplasty on the right side.

predominately bronchogenic. I do not believe that either of these ever exists in the pure form.

TUBERCULOSIS OF THE LARYNX

Tuberculosis of the larynx is always secondary to pulmonary tuberculosis. In all my experience, I never saw a case of laryngeal tuberculosis where a pulmonary lesion could not be demonstrated either clinically or by X-ray. From clinical observation it seems that the disease reaches the larynx chiefly by two routes: 1) Surface infection. This occurs mainly in the bronchogenic cases where the mucous membrane of the larynx is constantly bathed with bacilli-laden sputum, and infection occurs through direct contact. 2) Blood stream. This occurs usually in the hematogenous pulmonary cases where the larynx becomes involved by way of the blood stream, and the infection of the larynx is often severe and massive. Tuberculosis of the larynx therefore occurs in both bronchogenic and hematogenous cases, but its clinical manifestations differ immensely, and unless properly recognized and differentiated, we shall err greatly both in the prognosis and the treatment of this complication. In attempting to make a differential diagnosis in these two types of laryngeal infection, it is well to remember the following distinguishing points:

1.) In the bronchogenic tuberculosis, the onset is insidious. The patient usually consults his family physician or phthisiologist. The first symptoms are either referable to the lungs, such as cough and expectoration, or they may be constitutional, such as fever, asthenia, and loss of weight. In the hematogenous cases, the onset is

acute and the patient usually consults a laryngologist. The first symptoms are usually referable to the larynx.

2.) In bronchogenic cases, the examination of the chest often reveals physical signs indicative of pulmonary involvement. In the hematogenous cases, physical signs in the chest are usually scant and more often absent.

3.) In the bronchogenic cases, the sputum is often positive, while in the hematogenous cases, the sputum is usually negative, especially in the beginning. However, in the hematogenous cases, when ulcerations occur and the lesion begins to spread to other parts of the lungs bronchogenically, the sputum in these cases becomes positive, too.

4.) In the bronchogenic cases, we have characteristic X-ray findings showing infiltration of one or more of the lobes, often with cavitation. In the hematogenous cases, we have characteristic seeding involving either a portion of one or perhaps both lungs.

5.) In the bronchogenic cases, the first symptom is usually hoarseness or irritation in the larynx which necessitates frequent clearing of the throat. In the hematogenous cases, the patient usually begins with dysphagia and sometimes aphonia.

6.) In the bronchogenic cases, the laryngeal lesion is usually limited to a portion of the larynx, most often to the posterior commissure or to one or both vocal cords. In the hematogenous cases, the lesion is generally diffuse, and the epiglottis is invariably involved. The aryepiglottian folds and arytenoids are also frequently affected.

7.) In the bronchogenic cases, the larynx is usually involved alone, and the extension to other parts, such as tonsils, pharynx, and uvula, is rare; while in the hematogenous cases, the pharynx, soft palate, and the tonsils, the uvula and also other parts of the mouth are frequently involved, especially in the acute generalized miliary cases.

8.) In the bronchogenic cases, the prognosis, as far as the larynx is concerned, as a rule is good, and it is particularly favorable where collapse therapy is successfully applied. I have often seen cases of laryngeal tuberculosis clear up within a few weeks after an artificial pneumothorax has been instituted, even before a complete arrest of the lesion occurs. In the hematogenous cases, however, the prognosis depends largely upon the character of the infection. In the generalized miliary cases, where the larynx becomes involved acutely, the prognosis is invariably hopeless, and the life of the patient usually

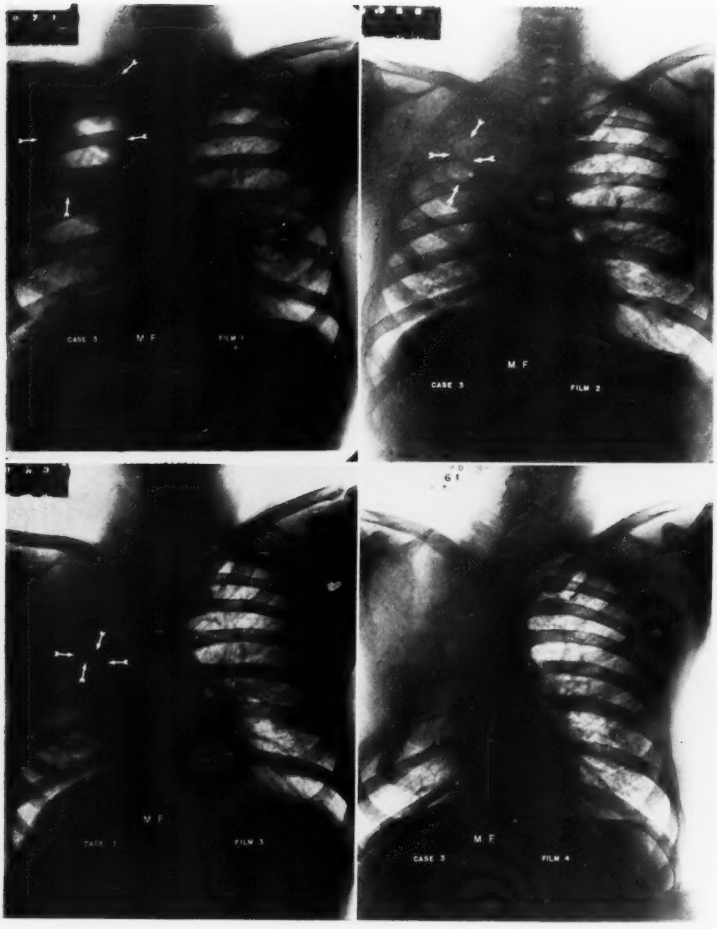


Fig. 6, Case 3.

Film I—Bilateral pulmonary tuberculosis with 12 cm. giant cavity in the right upper. Film II—75% reduction in cavity after Monaldi operation. Film III—Further reduction in cavity after first stage thoracoplasty. Film IV—Cavity no longer evident after second stage thoracoplasty.

extends from a few weeks to a few months, often terminating in meningitis. In the other hematogenous cases where the infection of the lungs is mild, the laryngeal lesion often clears, without much therapy of any sort.

9.) The laryngeal lesions in bronchogenic cases usually require little treatment. The larynx will clear with improvement of the lungs; while in the hematogenous, cases, treatment of the peracute larynx is of no avail, and here regardless of what you may do for the larynx, the lesion spreads like wild-fire and the patient dies from inanition. In these cases, I find that the best way to alleviate dysphagia is through the injection of the superior laryngeal nerves with 85% alcohol. Such injections often bring relief for several weeks and sometimes for months.

The following cases illustrate the beneficial effect of collapse therapy on tuberculosis of the larynx.

REPORT OF CASES

Case 1.—H. W., aged 29, was referred to me by Dr. Foster Murray of Brooklyn, N. Y., January 8, 1940. When first seen, the patient was completely aphonic. She stated that the aphonia came on November 23, 1939 and previous to that she was hoarse for some time. There was no history of dysphagia. The examination of the larynx showed a diffuse infiltration of both vocal cords with marked infiltration at the posterior commissure. The approximation of the cords was incomplete on phonation. There were ulcerations of both vocal cords (Fig. 1). The chest findings consisted of involvement of the right upper lobe with cavitations and considerable involvement of the left upper lobe also (Fig. 2, Case 1, Film 1). The patient was moderately toxic, and her weight was 64 kg. (141 pounds). The sputum was positive. Pneumothorax was induced on the right side with a gratifying result (Fig. 2, Case 1, Film 2). Symptoms became markedly diminished, temperature became normal and within a few weeks the voice began to clear up very rapidly. The sputum was now becoming negative occasionally, and during the past six months, her sputum has remained negative. On April 14, 1940, it was found necessary to perform a Jacobeus operation in order to improve the collapse on the right side. The result was very satisfactory (Fig. 2, Case 1, Film 3). As the pulmonary lesion improved, the voice cleared, and the larynx, on subsequent examinations, showed that both vocal cords were practically normal, the ulcerations healed, inflammation subsided, and the swelling at the posterior commissure markedly reduced (Fig. 1). In addition to collapse therapy on the right side, on December 3, 1940, it was also found necessary to induce a pneumothorax on the left side for the purpose of closing a rather well-defined cavity. In the meantime, because of rapid formation of adhesions on the right side, we were compelled to allow the lung on the right side to re-expand, and we are now continuing with the pneumothorax on the left.

Case 2.—F. S., aged 41, came under my care March 15, 1932. The original diagnosis was pulmonary tuberculosis, advanced and active, with extensive involve-

ment of the left lung and cavitation and early infiltration of the right apex. A pneumothorax was instituted on the left side in May 1933 with partial success. Phrenic exeresis was performed on the left side late in 1933 with good result. The lesion remained healed. In the meantime, the patient discontinued pneumothorax of his own accord. The lesion apparently remained healed for about four years, but in 1938 examination revealed formation of a large cavity in the right upper lobe (Fig. 4, Case 2, Film 1). A pneumothorax was attempted on the right side in July 1938 without success because of pleural adhesions. The cavity in the right lung, in the meantime, became larger until it had reached giant proportions. The larynx at this time also became involved with marked hoarseness and discomfort. Laryngoscopic examination showed infiltration of both vocal cords with ulcerations, infiltration at the posterior commissure (Fig. 3). April 15, 1940 a Monaldi operation was performed by Dr. Mark Williams on the right side. All the constitutional symptoms such as fever, cough, and expectoration became markedly diminished. The cavity was markedly reduced in size (Fig. 4, Case 2, Film 2) although the sputum still remained positive. Examination of the larynx May 6, 1941 shows that there is slight thickening at the epiglottis, both cords are completely healed and there remains only slight hyperplasia at the posterior commissure (Fig. 3). The voice is perfectly clear.

Summary: There was marked improvement of the larynx following the Monaldi operation.

Case 3.—M. F., aged 21, came under my care December 24, 1938. Initial examination disclosed far advanced pulmonary tuberculosis with giant cavitation in the right upper chest with infiltration extending into the mid-portion of the left lung (Fig. 6, Case 3, Film 1). The patient was very toxic and running fever of 100-102° in the afternoon. Examination of the larynx showed a small infiltration on the left side of the epiglottis, more on the dorsal surface. This seemed cystic. There was also an infiltration with superficial ulcerations of both vocal processes and hyperplasia at the posterior commissure (Fig. 5). The patient's voice was quite husky. A pneumothorax on the right side was attempted before the patient came under my care but was unsuccessful on account of adhesions. The pneumothorax was therefore abandoned. The patient was treated with bed rest for about a year. A phrenic operation was performed on the right side without much benefit. A Monaldi operation was performed January 5, 1940 with marked immediate improvement. The cavity became reduced in size 80-90% (Fig. 6, Case 3, Film 3), but since the sputum remained positive, this was followed by a first stage thoracoplasty, performed by Dr. Mark Williams on November 26, 1940 with a good result (Fig. 6, Case 3, Film 3). A second stage thoracoplasty was performed January 7, 1941 (Fig. 6, Case 3, Film 4). The cavity is now nearly obliterated although there is still a very small fistula at the site of the Monaldi. The patient's sputum has been negative right along, and generally he is markedly improved. Examination of the larynx shows both vocal cords are definitely healed, and there remains only a thickened epiglottis which I believe is cystic with slight hyperplasia at the posterior commissure which seems like scar tissue (Fig. 5). The voice is considerably clearer.

Summary: The larynx is markedly improved undoubtedly as a result of successful thoracic surgery.

RESUMÉ

1. Laryngeal tuberculosis is definitely on the decline. The incidence rate has dropped, from 25.6% in 1914, to 14.6% in 1934, to 3.6% in 1941.

The reasons for this are:

A. Improved case findings.

B. Improved methods of diagnosis.

C. Earlier treatment.

D. Increased employment of collapse therapy.

2. Laryngeal tuberculosis is always secondary to pulmonary tuberculosis, and the larynx becomes infected chiefly in two ways.

A. Surface infection. This usually occurs in the bronchogenic cases of pulmonary tuberculosis where the tubercle bacilli invade the larynx by penetrating the mucous membrane.

B. Blood stream. This form of invasion usually occurs in the hematogenous cases of pulmonary tuberculosis where the larynx often becomes involved simultaneously with the lungs.

We wish to thank Mr. Joseph Fobert of Liberty, N. Y., for his artistic drawings, Miss Gladys Tordik and the Camera Club of Otisville Sanatorium for their help in making the illustrations, and Dr. Gerald Pitegoff, for his assistance in collecting the data for this study.

7 LAW STREET.

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TABLE II.—440 OTISVILLE CASES

	Men		Women		Combined	
	235	53.4%	205	46.6%	440	100%
Clinical Diagnosis:						
Stage I	83	35.4%	97	47.3%	180	40.8%
Stage II	117	49.7%	75	36.5%	192	42.6%
Stage III	35	14.9%	33	16.2%	68	16.6%
	235	100%	205	100%	440	100%
Sputum:						
Positive	149	63.4%	138	67.3%	287	65.2%
Negative	86	36.6%	67	32.7%	153	34.8%
	235	100%	205	100%	440	100%
Larynx:						
Positive	6	3%	1	1%	7	2%
Negative	229	97%	204	99%	433	98%
	235	100%	205	100%	440	100%
Sputum in 7 Cases of Laryngopulmonary Tuberculosis:						
Positive	6	100%	1	100%	7	100%
Negative	0		0		0	
	6	100%	1	100%	7	100%
Therapy						
Routine	162	68.9%	112	54.6%	274	62.4%
Collapse	73	31.1%	93	45.4%	166	37.6%
	235	100%	205	100%	440	100%

TABLE III.—60 PRIVATE CASES

	Men		Women		Combined	
	32	53.3%	28	46.7%	60	100%
Clinical Diagnosis:						
Stage I	0	0	0	14.3%	4	6.6%
Stage II	8	25%	5	17.8%	13	21.7%
Stage III	24	75%	19	67.9%	43	71.7%
	32	100%	28	100%	60	100%
Sputum:						
Positive	23	71.8%	17	60.7%	40	66.6%
Negative	9	28.2%	11	39.3%	20	33.4%
	32	100%	28	100%	60	100%
Larynx:						
Positive	6	18.7%	5	17.8%	11	18.3%
Negative	26	81.3%	23	82.2%	0	81.7%
	32	100%	28	100%	11	100%
Sputum in 11 Cases of Laryngopulmonary Tuberculosis:						
Positive	6	100%	5	100%	11	100%
Negative	0		0		0	
	6	100%	5	100%	11	100%
Therapy:						
Routine	19	59.3%	12	42.9%	31	51.6%
Collapse	13	40.7%	16	57.1%	29	48.4%
	32	100%	28	100%	60	100%

TABLE IV.—500 CASES COMBINED

	Men		Women		Combined	
	267	53.4%	233	46.6%	500	100%
Clinical Diagnosis:						
Stage I	83	31.1%	101	42.3%	184	36.8%
Stage II	126	46.8%	80	34.3%	205	41 %
Stage III	59	22.1%	52	23.4%	111	22.2%
	267	100%	233	100%	500	100%
Sputum:						
Positive	172	64.4%	155	66.5%	327	65.4%
Negative	95	35.6%	78	33.5%	173	34.6%
	267	100%	233	100%	500	100%
Larynx:						
Positive	12	4.4%	6	3%	18	3.6%
Negative	255	95.6%	227	97%	482	96.4%
	267	100%	233	100%	500	100%
Sputum in 18 Cases of Laryngopulmonary Tuberculosis:						
Positive	12	100%	6	100%	18	100%
Negative						
	12	100%	6	100%	18	100%
Therapy:						
Routine	181	67.7%	124	53.1%	305	61%
Collapse	86	32.3%	109	46.9%	195	39%
	267	100%	233	100%	500	100%

TABLE V.—COMPARATIVE ANALYSIS OF 500 CASES OF PULMONARY TUBERCULOSIS

	1914		1934		1941	
Clinical Diagnosis:						
Stage I	124	24.9%	113	22.6%	184	36.8%
Stage II	303	60.6%	274	64.6%	205	41 %
Stage III	73	14.6%	113	22.6%	111	22.2%
	500	100%	500	100%	500	100%
Sputum in 470 Pulmonary Cases:			500 Cases		500 Cases	
Positive	332	70.6%	362	72.4%	327	65.4%
Negative	138	29.4%	138	27.6%	173	34.6%
	470	100%	500	100%	500	100%
Larynx:						
Positive	128	25.6%	73	14.6%	18	3.6%
Negative	373	74.4%	427	85.4%	482	96.4%
	500	100%	500	100%	500	100%
Sputum in 128 Laryngopulmonary Cases:			73 Cases			
Positive	116	91%	72	98.6%	18	100%
Negative	12	9%	1	1.4%	0	
	128	100%	73	100%	18	100%
Treatment:						
Routine	500	100%	400	80%	305	61%
Collapse			100	20%	195	39%
	500	100%	500	100%	500	100%

LIV

PARALYSIS OF THE LARYNX*

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and

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Paralysis of a vocal cord usually is secondary to a lesion of a peripheral nerve. Central lesions account for about 10% of cases. Peripheral lesions may involve the vagus nerve at the jugular foramen, the superior laryngeal branch in the neck and the recurrent or inferior laryngeal branch in the neck or mediastinum. While the causes are numerous there is common agreement that malignant growths exclusive of laryngeal neoplasms, trauma to a nerve in the course of surgical treatment of disease of the thyroid gland and aneurysm of the aorta are the most frequent. Agreement, however, is not unanimous on the innervation of the larynx, the position assumed by the vocal cord immediately following injury to the recurrent nerve and the changes that occur subsequently depending on whether there is complete and permanent loss or restoration of laryngeal function. In this paper an attempt will be made to correlate the experimental and anatomical findings of certain investigators with clinical observations made in a group of over 250 cases of laryngeal paralysis of peripheral origin.

The views expressed by Ónodi regarding the nerve supply of the larynx are generally accepted, namely, that all of the muscles of the larynx except the cricothyroideus are innervated unilaterally by the corresponding recurrent laryngeal nerve. The arytenoideus, an unpaired, midline muscle, is supplied by both right and left recurrent nerves. The cricothyroideus is supplied by the corresponding external subdivision of the superior laryngeal nerve. Other investigators believe that certain muscles receive an additional nerve supply from other fibres. Luschka believes that the arytenoideus receives fibres from the superior laryngeal nerve. In a number of textbooks it is stated that the superior laryngeal nerve may supply

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fibres to the thyroarytenoideus as well as the abductor and adductor muscles, while the recurrent nerve may send filaments to the cricothyroideus.

Because of the apparent differences of opinion regarding the nerve supply of the larynx, Lemere reëxamined the entire subject of laryngeal muscle innervation. His work constitutes the most exhaustive and complete study of this problem that has been done thus far and should bear great weight in any clinical discussion of laryngeal paralysis. Lemere is in entire agreement with the views expressed by Ónodi. Neither he nor any other worker in this field has ever produced contraction of any of the laryngeal muscles except the cricothyroideus by stimulation of the superior laryngeal nerve. He found that the branches of the internal subdivision of the superior laryngeal nerve which appear to supply the arytenoideus in reality pass through this muscle to supply the overlying mucosa. While it has been suggested that nerve impulses might reach the laryngeal muscles by routes other than those described none have been discovered. A median or third laryngeal nerve is present in certain experimental animals, notably the dog, but none has been demonstrated in man. Lemere also redirected attention to a function of the cricothyroideus which often is overlooked, namely, its action as an adductor. This exerts a definite influence on the position of the vocal cords in recurrent nerve paralysis.

While multiple innervation of the laryngeal muscles and anomalous distribution of nerves may suffice to explain certain clinical observations in laryngeal paralysis, these have not been substantiated by experimental or anatomical study. Adherence to such views merely serves to confuse rather than clarify the questions under consideration.

The terminology applied to the position of the paralyzed vocal cord also appears complicated. References are made to median, paramedian, mid, adductor, phonatory, intermediate and cadaveric positions. The median, phonatory and adductor positions should be considered as synonymous and are interpreted as referring to a vocal cord in the midline. A cord in a paramedian position is adjacent to the midline. The tension of the vocal cord occupying one of these positions usually is normal, there are no voice changes and there is little or no dyspnea unless the paralysis is bilateral. In the mid and the intermediate positions the cord is approximately midway between abduction and adduction. There usually is some relaxation of the cord. If there is compensatory movement of the normal cord be-

yond the midline, voice disturbances are not marked. If the paralysis is bilateral there usually is marked hoarseness. This position is described by some writers as the cadaveric. Others consider the true cadaveric position as that assumed when there is complete paralysis of all the muscles with complete muscular relaxation. This occurs when the superior and recurrent nerves are sectioned or their continuity interrupted by disease as often is observed in the jugular foramen syndrome. In the true cadaveric position the arytenoid occupies an intermediate or midposition and is tilted forward; the vocal cord is relaxed and flaccid, occupies a lower level and often appears narrower than normal. The edge of the membranous cord is bow-shaped with the concavity directed inward; the tip of the vocal process of the arytenoid is directed inward and appears as a definite prominence. In bilateral paralysis the membranous glottis is ellipsoid while the cartilaginous glottis is triangular in shape. The voice is husky; there is air waste and cough is difficult owing to inability momentarily to close the laryngeal airway. We always have considered this as a permanent paralysis in contradistinction to that in which the cord is somewhat abducted and relaxed, but in which restoration of function is observed or the cord assumes a median position.

It has been shown experimentally and observed clinically that injury of a recurrent nerve produces paralysis of all of the muscles on that side of the larynx with exception of the cricothyroideus supplied by the superior laryngeal nerve and the arytenoideus which receives its innervation from both recurrent nerves. This constitutes unilateral abductor or posticus paralysis. What is the position of the vocal cord? There should be loss of abduction of the corresponding vocal cord which may occupy a position anywhere from the midline to a point near the intermediate position. If near the midline the tension usually is good, the voice normal or but slightly disturbed and the arytenoid practically in normal position. If, however, the cord is near the intermediate position there is definite forward tilting of the arytenoid with loss of tension. Voice disturbances are marked until the opposite vocal cord compensates by crossing the midline during phonation. This should not be considered as the true cadaveric position, for function of the cricothyroideus and arytenoideus is unimpaired. In a considerable number of cases of unilateral recurrent paralysis occurring after thyroidectomy and in three cases following surgical extirpation of a pharyngeal diverticulum, all of which were observed within a few days to one week following operation, the paralyzed cord was found partially adducted with loss

of tension. There was some hoarseness. By some observers this was considered as a cadaveric position. In the three postdiverticulectomy cases function of the larynx was found normal when examined seven weeks to four months after operation. In a few of the cases of paralysis following thyroidectomy normal function returned, while in the larger group the vocal cord assumed a median or paramedian position. In a case of injury to the esophagus following the ingestion of lye there developed during the course of treatment paralysis of the left vocal cord. The cord was partially adducted with some loss of tension; there was forward tilting of the arytenoid and hoarseness. Within three weeks improvement of the voice was noted, and the cord had assumed a median position with good tension. In a case of bilateral paralysis complicating carcinoma of the trachea the cords were immobile in a partially adducted position with some loss of tension, marked hoarseness and no dyspnea. There was no change in the position of the cords up to four days before death.

Were the vocal cords in these cases in a true cadaveric position? We do not believe so. In his experimental studies on the effects of nerve sectioning in dogs, Lemere found that bilateral section of both recurrent nerves "produces a partially adducted position of the glottis which is midway between full adduction and the cadaveric position that has always previously been associated with this type of paralysis." The cadaveric position is assumed only after injury to both recurrent and superior laryngeal nerves or it may also be observed after atrophy of the cricothyroideus in conjunction with recurrent nerve paralysis.

The degree of adduction and the occurrence of the cadaveric position vary greatly in the experience of many observers. In a study of 235 cases of paralysis of the recurrent nerve by Smith, Lambert and Wallace, the position of the paralyzed cord was recorded in 117 cases. On the first examination it was most commonly found in the cadaveric position. According to New and Childrey the paralyzed cord is found in one of two positions, the median line or the cadaveric. In a report of 217 cases of paralysis of which approximately 190 were of peripheral origin they found the paralyzed cord in the median line in about 90% of the cases of recurrent nerve involvement. In a small percentage of cases the cord was found in the cadaveric position. In the series of 270 cases of paralysis reported by Suehs the position of the vocal cord was stated in 230. Of these the cord was in the median line in 131 and in the so-called cadaveric position in 99. There was some loss of tension in 146 cases.

The cause for this variation in position as well as disturbance in tension occurring promptly after injury to the recurrent nerve cannot be satisfactorily explained. In a discussion of the laryngoscopic findings in unilateral paralysis, Irwin Moore stated that in the first stage the vocal cord is immobile during ordinary respiration and resting close to the middle line of the larynx, while on deep inspiration the paralyzed cord fails to abduct beyond the cadaveric position. Recently we observed the position of the vocal cords of patients during laryngectomy performed under local anesthesia after section of the superior laryngeal nerves and then following section of the recurrent laryngeal nerves. After the superior nerves are cut there is no apparent change in the appearances of the cords. Although the cords meet in the midline on phonation, usually no sound can be p.oduced; cough, however, seems unimpaired. On section of a recurrent nerve the corresponding cord immediately assumes the median position. Obviously the period of observation was brief and the subsequent behavior of the cord could not be determined. On the other hand cases of unilateral paralysis following thyroidectomy have been observed within 24 hours after the occurrence of paralysis. The paralyzed cord was partially adducted and there was loss of tension. Is the immediate effect of section of a recurrent nerve complete loss of abduction with full adduction and later is there some relaxation of the power of the adductors? The finding of a paralyzed vocal cord partially adducted with loss of tension in eight to fourteen days after thyroidectomy has been a common experience. Since a majority of the paralyzed cords which do not recover normal function ultimately assume a median position it does not appear logical that the original paralytic position was cadaveric with complete muscular relaxation.

What is the fate of the paralyzed cord? A certain number regain normal function as was observed in three cases of paralysis following diverticulectomy, four cases of unilateral paralysis following thyroidectomy, six in which the cause of the paralysis could not be determined and several which were considered as being luetic in origin. In addition one case of bilateral paralysis following thyroidectomy regained normal function of the left cord, the right assuming a median position. Recovery commonly occurs within three or four months although longer periods have been reported. Recovery in one case was observed within five weeks following thyroidectomy. Turner states that although the division of unilateral paralysis of the recurrent nerve into three stages appears to be confirmed by experiments on animals, and is usually accepted, many observers never

have seen a case of incomplete paralysis pass on to the complete stage. New and Childrey found the paralyzed cord that does not return to function pulls to the median line. They have not observed a case in which the cord changed from the midline to the cadaveric position. That has been my experience and coincides with Suehs' findings, although in one patient observed 14 years following thyroidectomy the right vocal cord was found in a true cadaveric position. The voice changes were of but two and a half years' duration. This could be explained on the basis of atrophy of the right cricothyroideus muscle with consequent failure of tension and adduction. While data on follow-up examinations is limited there have been observed 19 cases of paralysis following thyroidectomy performed from many months to 21 years previously. In 18 of these the cords were in the median or paramedian position. In the remaining case described above the cord appeared in a true cadaveric position.

Return of tension to the paralyzed cord which assumes a median position, if function is not restored, apparently is the rule in postthyroidectomy cases. This commonly occurs within a few months after onset. In one patient with paralysis and hoarseness following thyroidectomy a partially adducted vocal cord with loss of tension regained its tension and assumed a median position with normal phonatory function 17 days after operation. Persistence of the paralyzed cord in a partially adducted position with loss of tension has been observed more often in cases due to aneurysm of the aorta and in carcinoma, especially of the esophagus.

SUMMARY

On the basis of personal clinical observations and experimental and anatomical studies of other observers, it is apparent that injury to a recurrent laryngeal nerve results in partial adduction with loss of tension of the corresponding vocal cord. It is possible that in man this is preceded by a brief period of full adduction. If recovery of function does not occur the paralyzed cord assumes a median position, tension improves and the voice returns to normal. A vocal cord in the midline does not later assume a cadaveric position.

The true cadaveric position is observed infrequently in recurrent paralysis unassociated with loss of function of the corresponding superior laryngeal nerve. The occasional cadaveric cord observed in uncomplicated recurrent paralysis probably results from atrophy of the cricothyroideus.

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LV

UNUSUAL POSITION OF THE RIGHT RECURRENT LARYNGEAL NERVE

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Of the many anomalies that occur in embryological development, one of the most interesting is that of the anomalous origin of the right subclavian artery resulting in an unusual course of the right recurrent laryngeal nerve. Normally this nerve loops around the right subclavian artery before it starts its upward course. As early as 1823 Stedman[1] demonstrated in a female cadaver, nerves that arose from the right vagus nerve passing directly to the larynx. Regarding these nerves he stated, "It is probable that, in this case, the nerves coming directly from the trunk of the vagus, on the right side, performed the same function as the recurrent; for they were distributed very much in the same manner as the branches of that nerve." The right subclavian artery arose on the aortic arch to the left of the left subclavian artery and passed posterior to the esophagus and the trachea and thence to its normal position in relation to the scaleni muscles.

Since this time this anomaly has been described on several occasions. Hart[2] in 1826 reports in detail such a case. In a footnote of the Guy's Hospital Reports in 1837, John Hilton[3] wrote, "In our dissecting-room, last winter, we had a subject, in which the right subclavian artery came off from the posterior part and the left side of the transverse portion of the aorta. In that case, the right recurrent nerve did not curve around the artery, but was detached from the pneumogastric at an acute angle with the descending part of the nerve, about opposite the fifth cervical vertebra."

Herand as quoted by Hooper[4] presented a specimen at the Anatomical Society of Paris in 1846 showing a high origin of the right recurrent laryngeal nerve but did not mention the anomalous vessels. Demarquay in 1848, as quoted by the same author,[4] on the contrary, demonstrated a specimen of irregular origin of the right subclavian artery with "absence" of the recurrent nerve on the same side.

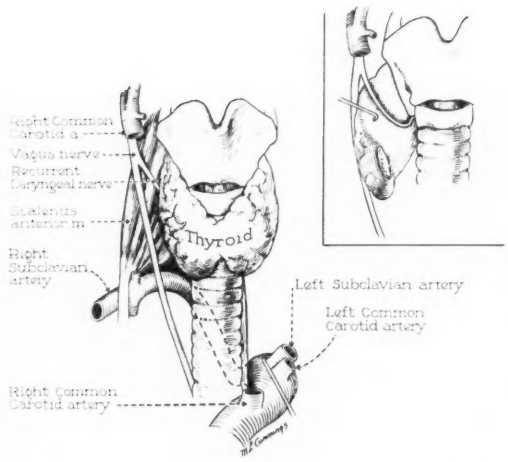

Fig. 1. The position and course of the right recurrent laryngeal nerve are clearly shown. Note the deep notch formed on the posterior surface of the thyroid gland by the nerve as it passes almost in a direct horizontal course to the larynx and the position of the right subclavian artery posterior to the esophagus and trachea.

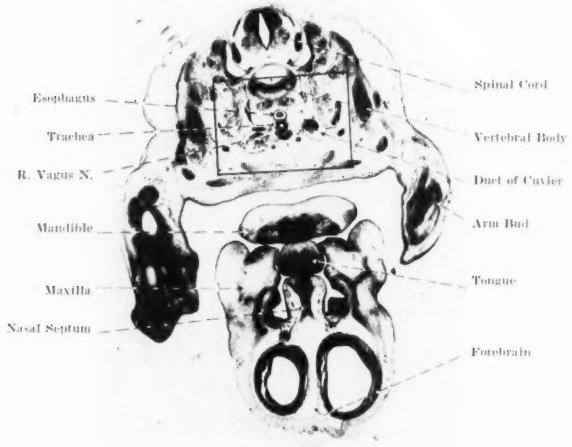

Fig. 2. Transverse section 17 mm. human embryo, x's 9. Subsequent high power sections are indicated by rectangle. Note relative size of upper trunk, head, and arm buds, and the amount of flexion of head.

Hooper[1] in 1887 reported a case of Professor Dwight's at the Harvard Medical School dissecting room of both the anomalous development of the right subclavian artery and the concomitant high position of the right recurrent laryngeal nerve.

More recently, in 1924, Milianitch[5] pointed out such anomalies. During the course of a subtotal thyroidectomy in 1932 Pemberton and Beaver[6] encountered a high position of the right recurrent laryngeal nerve and presented a suspected origin of the right subclavian artery directly from the arch of the aorta to the right of the midline and to the right of the right common carotid artery.

In 1939, at the Anatomical Laboratories of the University of Michigan, a cadaver was found that presented the aforementioned anomalies. Figure 1 shows an artist's reproduction of the specimen. The right recurrent laryngeal nerve passed directly to the larynx by a single trunk. On the posterior surface of the thyroid gland a deep notch was formed through which the nerve passed, becoming intimately associated with the capsule of the gland. The position of the great vessels and particularly the right subclavian artery is shown clearly.

Embryologically the recurrent laryngeal nerves are associated early with the development of the large vessels of the thorax. Of the six paired primitive aortic arches the fourth on each side remains normally, the right persisting as the right subclavian artery and the left remaining as the aortic arch. The right recurrent nerve is impinged beneath the right subclavian artery, and the left is impinged beneath the aortic arch. As the embryo elongates and the thoracic organs descend, the nerves assume their recurrent course. In order to present this process as it actually occurs, sections of human embryos, from the Huber and Patten collection, were reviewed. Figures 2 to 6 are photomicrographs of a 17 mm. human embryo taken from the Huber collection. It will be noted that even in such a young embryo the right recurrent nerve has already assumed a recurrent course.

To account for the unusual position of the right recurrent nerve, as shown in Fig. 1, we must turn to the anomalous development of the great vessels of the thorax. When Stedman[1] first presented his case he commented, "Nature seems to have been forced to have recourse to this arrangement, from the singular situation of the subclavian." Hart[2] elaborates still further on his case, for he recognizes and suggests an anatomical embryological disturbance as a basis

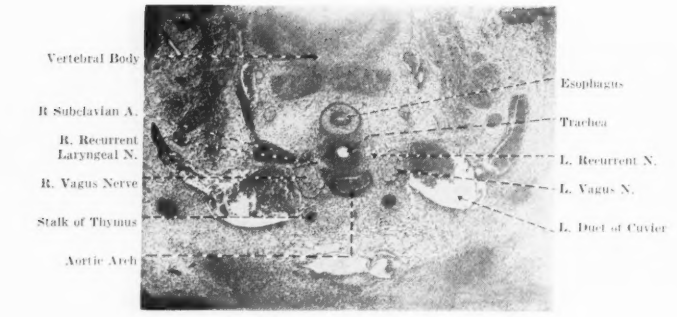

Fig. 3. High power section, x's 39, of region marked in rectangle Fig. 2. The right recurrent laryngeal nerve is shown as it arises from the right vagus nerve directly beneath the right subclavian artery. The very top of the aortic arch is shown cut in tangential section. The left recurrent laryngeal nerve has already arisen from the left vagus nerve and is in a recurrent position at this level.

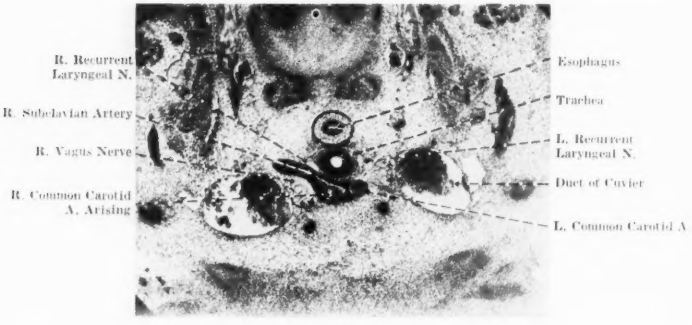

Fig. 4. Section through similar area as previous plate, x's 39, but more cephalad. The right recurrent laryngeal nerve is seen emerging from the under surface of the right subclavian artery and is just starting its recurrent course. The right innominate artery has already formed the right subclavian artery and the right common carotid artery. The left common carotid artery is shown just after it has arisen.

for the anomaly. Hooper[4] clearly explains the process by which such a disturbance takes place.

Probably the most recent clear explanation is given by Piersol.[7] He lists five different groups of anatomical embryological variations of the great vessels of the thorax. The group that particularly concerns us is Group II, and of this group he states, "A more frequent anomaly is the complete persistence of the distal portion of the right aortic arch associated with the disappearance of a greater or less portion of its proximal part, the result being the apparent origin of the right subclavian artery from the descending aorta, whence it passes to the right behind the trachea and the esophagus." With failure of development of the proximal portion of the right fourth primitive aortic arch, no vessel remains in contact with the developing right recurrent laryngeal nerve, and instead of being dragged down in its usual recurrent position it continues its unusual course to the larynx.

Of many anomalous patterns of the great vessels of the thorax the position of the recurrent nerves vary with these patterns only when the nerve-vessel relationship is disturbed. Thus in Piersol's Group III the right fourth primitive aortic arch persists as the aortic arch; the proximal portion of the left fourth primitive aortic arch remains as the left subclavian artery. This is not, as Piersol points out, a true *situs viscerum inversus* but simply a reversal of the position of the aorta and its vessels. We would expect the recurrent nerves to be in a recurrent position in such a case but looped about their respective vessels in just the reverse order of the normal. Brenner, as quoted by Hooper,[4] reported a case wherein the right vertebral artery represented the fourth primitive aortic arch on the right and the right recurrent laryngeal nerve "branched from the pneumogastric opposite the sixth cervical vertebra and turned around the vertebral artery to reascend to the larynx."

In the case presented by Pemberton and Beaver[6] it appears from their illustration that their suspected anomaly would fall in Piersol's Group V. Although he mentions no position of the recurrent nerves associated with these anomalies, it is apparent that as long as the proximal portions of the primitive fourth aortic arches persist, the nerves would assume a recurrent position, and only with failure of development of the proximal portion of the arches would the nerve be found in a high position. As stated previously, Piersol shows that with failure of development of the proximal portion of the right fourth primitive aortic arch such an anomaly would fall in his Group

Fig. 5. Section through same area, x's 39, still more cephalad. The recurrent course of the recurrent laryngeal nerves is shown clearly in this section. Note comparative large size of the nervous tissue structures, especially the comparative sizes of the recurrent nerves and vagi to the common carotid arteries, esophagus, and trachea.

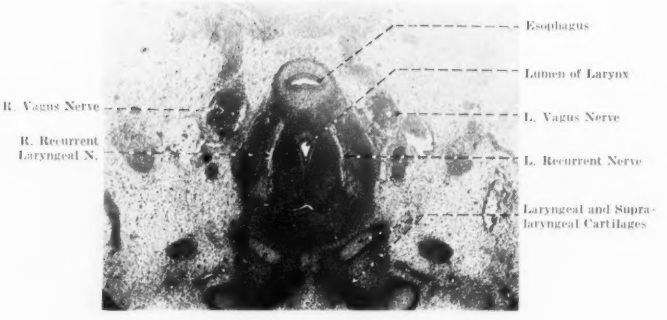

Fig. 6. Section still more cephalad, x's 39, through the larynx showing the recurrent nerves spreading out into the substance of the larynx.

II and coincide with Fig. 1. We know what happens to the right recurrent laryngeal nerve in such instances. It becomes apparent that the suspected anomaly as shown in the figure presented by Pemberton and Beaver might well have been an anomaly such as presented here.

CONCLUSIONS AND COMMENTS

1. A case of an anomalous origin of the right subclavian artery resulting in an unusual position of the right recurrent laryngeal nerve is presented.

2. The normal position of the recurrent nerves is dependent on the normal development of the proximal portions of each fourth primitive aortic arch, and disturbances of this nerve-vessel relationship will result in an unusual position of the recurrent nerve.

3. Although this anomaly occurs infrequently, surgeons working in the neck must be cognizant of it, if resultant vocal cord paralysis is to be avoided.

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LVI

LARYNGEAL TUMORS — VOICE RECORDINGS BEFORE AND AFTER OPERATION*

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ATLANTA

The voice-recording machine is the new ally of the X-ray, still photography and motion pictures in surgical expositions. The "before and after" recordings form a permanent record for the files of the laryngologists, just as the clinician has the thermometer and the findings of the sphygmomanometer as a record, the urologist has the kidney function test and the plastic surgeon has pictures taken of his patients before and after operations. In the same way, in the recording of laryngeal tumors, the "before and after" records are of inestimable value to case histories.

Voice recordings before and after operation were auditioned to those present illustrating the three most commonly used laryngeal operations, such as the simple removal of papillomas with the Lynch suspension apparatus, the complete resection of the vocal cord for carcinoma, and the use of an artificial larynx following total laryngectomy for carcinoma.

Hoarseness points directly to disease or dysfunction of the larynx. It occurs only when the tension, vibration, or approximation of the vocal cords is disturbed. Chevalier Jackson states that the whispered voice is never lost as long as air can pass through the glottis. Hoarseness that persists only a few days is a frequent complication of the common cold and does not need the treatment of a specialist: it will clear up when the patient stops coughing. Hoarseness, however, that persists more than two weeks should be thoroughly investigated. This investigation should include a general examination with an X-ray of the chest, for it may result from pressure on the recurrent laryngeal nerve as by lung cancer or aneurysm of the arch of the aorta. Usually, however, hoarseness is caused by intrinsic disease of the larynx, and therefore a careful ex-

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amination of this organ should be made in every case. This should usually include direct inspection by means of a laryngoscope and in case of any doubt a biopsy.

The fundamental cause, the *Ursache,* of such disease is unwise use of the voice. It may be that when public speakers take lessons in voice production too great strain on the vocal cords will become uncommon. I understand that a course in radio speaking will be instituted in the Baptist Theological Seminary this fall in order to increase the ministers' sphere of influence and also decrease the strain on their voices when addressing large audiences, thus making them more useful. It may be too that the increasing use of the lapel microphone will enable public speakers to make themselves heard without taxing their vocal cords. However, it should be noted that about 10% of laryngeal affections do not originate in the cords.

Abuse of the voice is not the only cause of disease of the larynx. Infection, chronic inflammation or irritation from watever source have been held responsible, as well as, of course, tobacco and alcohol. Tobacco and alcohol may have been incriminated only because laryngeal lesions are more than twice as common in men as in women, and in a former day women did not to any extent indulge in public speaking, tobacco or alcohol. However, on account of the increased use of tobacco and alcohol by women in all probability in future years there will be an increase in laryngeal affections in women.

In addition to hoarseness, cough, dysphagia, hemoptysis, stridor, dyspnea, cyanosis, asphyxia, a sense of fullness in the throat, gagging and retching are symptoms of disease of the larynx, although disease may be present without any of these symptoms.

The etiologic factors named seem to play a role in all diseases of the larynx. These diseases may be roughly classified under three heads:

1. Inflammatory lesions.
2. Benign neoplasms.
3. Cancer.

Although laryngeal cancer accounts for about 5% of all carcinomas, it cannot be considered in detail here, as this paper will deal more specifically with tumors of the benign type.

Chronic inflammatory diseases of the larynx and actual neoplasms are usually linked together in a discussion of benign tumors of the larynx. The granulomas account for somewhat more than half, and we shall begin with them.

First in importance is tuberculosis. Tuberculosis is never primary in the larynx, and both physical examination and the X-ray will give evidence of far-advanced pulmonary phthisis. The sputum will be positive, and direct smear from the larynx will also show the *mycobacterium tuberculosis*. Occasionally tuberculous granulation tissue must be distinguished from papillomas. Pain in swallowing is a frequent serious complication. The most important thing in the treatment of this condition is of course general care for the systemic infection together with a regimen of absolute silence. Opiates are contraindicated. Careful cauterization is often employed. Lukens has advised the application of chaulmoogra oil in liquid petrolatum. In some cases tracheotomy may be advisable. The prognosis is grave.

Syphilis of the larynx is much more apt to be mistaken for cancer than for a benign neoplasm. It also requires judicious and long continued treatment of the systemic infection. There is of course no guarantee that the organisms of tuberculosis and of syphilis may not both invade the same larynx, and this larynx may even be the seat of a tumor.

Granulomas are covered by a thin layer of squamous epithelium which is often lost when prepared for microscopic examination. Early granulomas are mostly inflammatory tissue but fibrosis increases as the condition becomes more chronic. Since the presence of a granuloma does not exclude the presence of a neoplasm, it is most important to examine the larynx in detail at the time of removing the granuloma in order to rule out this possibility.

Contact ulcers are not infrequently associated with granulomas. An ulcer not only causes pain but increases hoarseness. Contact ulcers must be distinguished from tuberculosis and epithelioma. The treatment of such ulcers requires the removal of the granuloma and the ulcer with complete silence until the wound has healed.

The incidence of benign neoplasms of the larynx cannot be estimated, for relatively few of them are ever seen by a laryngologist. It is most difficult to classify them satisfactorily for even the pathologists have not yet been able to come to agreement as to their classification.

Papillomas account for about one in four of all tumors of the larynx. Some may be congenital but more probably result from inflammation. Chevalier Jackson has noted 28 cases in men who had been gassed in the World War. Papillomas may originate elsewhere to involve the vocal cords later or may spread widely and

rapidly from the cords. They are by far the most common chronic disease of the larynx in young children. Pure chronic inflammatory lesions at this age are almost unknown. Papillomas are often self-limited and may clear up spontaneously about the time of puberty. In adults papillomas are only too apt to continue to grow, and recurrence after removal is often encountered at any age.

The treatment of papillomas is not satisfactory as is evidenced by the many methods advocated: excision, electrocoagulation, cauterization, escharotics, radiation by radium and roentgen rays, thyrotomy, tracheotomy and internal medication. Excision of the papillomas flush with the surface without injury to the underlying tissue is the best treatment in children. Electrocoagulation is often used with a carefully controlled current. This operation is bloodless and allows for better operating procedures. Neither the actual cautery nor an escharotic should be used, for either may damage normal tissue so much as to make it impossible to restore the normal voice. Radium works well, but the resultant scarring of the larynx constitutes a very decided disadvantage to its use. Roentgen radiation has been generally unsatisfactory, but some workers have found it to be satisfactory if given in proper and sufficient dosage. Thyrotomy should be reserved for cases in which malignancy is suspected. Tracheotomy may be necessary in cases where the laryngeal obstruction is great. If tracheotomy is resorted to, the cannula and wound should be kept clean or there will be a spread to the trachea and bronchi. Internal medication is futile.

Fibromas may be polypoid or sessile, highly vascular or mainly fibrous. They may occur anywhere, arising from the perichondrium, mucosa or the tissue between. They vary considerably in size and rank second only to papillomas in frequency. New and Erich have reported one case of a neurofibroma in a woman of 49 years without any other evidence of von Recklinghausen's disease.

Vocal nodules are most common in the throats of improperly trained singers and others who use their voices professionally, such as school teachers. Often they are bilaterally symmetrical. The most important thing in treatment of "singer's nodes" is proper training of the voice, for if the etiologic factor is not eliminated they will recur after removal and the surgeon may even be sued for the impaired voice.

Hematoma, an area of localized hemorrhage in a vocal cord, is produced by short violent strain of the voice. Unless the blood is promptly evacuated, a hematoma will usually undergo organization,

resulting in persistent tumor of the cord which may be mistaken for a fibro-angioma. After organization the hematoma may have to be removed surgically.

The various types of angiomas are of pathologic interest but clinically they are rather rare. They have often been considered of congenital origin, but the large majority are found in adults. The occasional hemangioma found in the infant is usually associated with hemangiomas in other parts of the body. The cavernous or capillary hemangiomas are of particular interest. They vary in size from 2 to 30 mm. and are apt to be pedunculated. Being composed of erectile tissue they may be definitely diagnosed by the phonation test: that is, these tumors become erect, firm and of deeper color on phonation.

Teratomas are also rather rare in the larynx, but there is a tendency for them to become malignant.

Myomas, myxomas, lipomas, adenomas, chondromas, osteomas, amyloid and thyroid tumors, xanthomas, and gouty tophi can only be mentioned. Many an experienced laryngologist has never seen most of them.

Pachydermia is an epithelial thickening of the mucosa of the larynx. Keratosis is a localized piling up of cornified epithelium on the cords. The tumor appears as a white mass projecting from the free end. There is usually an associated chronic inflammation of the mucosa. Epithelial hyperplasia and leucoplakia are uncommon, but they are important because of their potential malignancy. Leucoplakia is confined to the true vocal cords and appears as whitish patches which are thick with a papillary appearance. Hyperplasia varies in its appearance—it may be nodular, polypoid, papillomatous, or it may resemble leucoplakia.

Cysts, laryngocele, eversion of the ventricle, prolapse of the sacculus, although not true tumors present problems in differential diagnosis.

Cysts of the larynx occur infrequently. They average 3 to 5 mm. in diameter but they may become as large as 4 cm. Unlike other tumors of the larynx they are about equally divided between the two sexes. They have been classified into three types:

1. Retention cyst.
2. Pseudocyst.
3. Connective tissue cyst.

While one may be tempted to puncture the cyst, for permanent relief he should excise it and cauterize the base.

In 1937 I reported the case of a young clergyman who had been annoyed with a difficulty in breathing. When he would reach the climax of a sermon and his voice was raised with emotional fervor, he would suddenly lose his voice entirely and become short of breath. When he would sit down and take a swallow of water he had the sensation of swallowing something solid along with the water. Then he could talk again and was free of dyspnea. Indirect examination had been performed repeatedly, at times revealing nothing, at others a mass about the size of a pecan. A cyst 2 cm. in diameter with a pedicle 2.5 cm. was removed, and the patient was entirely relieved of his occupational disability.

Laryngocele is a very rare anomalous condition, but it may be so easily mistaken for a tumor in appearance that it should be mentioned. Diagnosis is made by enlargement of the protrusion on blowing or coughing, or enlargement of the intrusion by pressure from without. If there is endolaryngeal intrusion, the sac should be removed to prevent possible dyspnea and asphyxia.

Eversion of the ventricle appears as a sessile tumor projecting from the ventricle without any line of demarcation between the tumor and ventricular band. If the tumor is manipulated by direct laryngoscopy, it is easy to determine the character and to realize one is dealing with a prolapse. The treatment is return of the eversion by direct laryngoscopy. Often if one clips out part of the mucosa for biopsy, the condition will be cured. Conservative treatment in the removal of tissue is best. Any associated condition should be treated. Permanently impaired voice may result.

COMMENT

When benign tumors of the larynx interfere with speaking, when they produce obstructive dyspnea or localized irritation, or when they are apt to cause such symptoms, or of course when they are of a precancerous nature, they require surgical removal. The greatest care should be exercised to leave the normal tissue unimpaired so far as possible: in contrast to the treatment of malignant lesions, as little normal tissue as possible should be removed with the benign tumor. The removal may be made with a knife, with a biting forceps or with a cautery.

The most important reason for the removal of benign tumors of the larynx is, of course, voice preservation; following this, to re-

lieve increasing obstructive dyspnea which impairs health and may gradually, through the growth of the tumor, or rapidly, through an inflammatory process, produce strangulation. The sudden onset of asphyxia is due to a sudden inflammatory swelling of the tumor. Finally, in cases of affections of the larynx, may I emphasize the responsibility of the surgeon, for the proper treatment depends on the diagnosis: on the surgeon is placed the responsibility not only for the future voice of the patient but for his happiness and even for his very life.

PONCE DE LEON EYE, EAR AND THROAT INFIRMARY.

LVII

THE PATHOLOGIC ANATOMY OF POLYPS OF THE LARYNX*

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AND

WALTER H. SEGALL, M.D.

CHICAGO

It does not seem to be generally appreciated that the most common benign laryngeal tumor is a polypoid growth originating on the vocal cord margin. The anatomical concepts underlying this recognition were first advanced by Reinke,[1] Hajek,[2] and Chiari.[3] A recent analysis of a large series of benign tumors of the larynx by New and Erich called attention to the preponderance of non-neoplastic growths.[4] In this series the so-called "inflammatory tumors," which included polypoid types, predominated. It is the purpose of this paper to clarify further the clinical pathology of these polypoid growths.

There has been considerable variation in the nomenclature applied to vocal cord growths. They are most frequently designated as fibromas,[5] occasionally as angiomas, fibro-angiomas, myxomas and lymphangiomas. Whereas it is quite true that fibromas, fibro-angiomas and the like may occur, they are relatively uncommon, as are all true benign laryngeal tumors with the exception of papillomas.[4]

Waldapfel has recently reviewed the contributions of Reinke and Hajek and has presented additional evidence in support of the all-important role of the subepithelial space in pathologic conditions of the vocal cords.[6]

ANATOMY

For a proper understanding of this subject is is desirable to re-emphasize briefly its anatomical aspects. Reinke's space sur-

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rounds the free margin of the vocal cord and is limited in all directions. It extends from near the anterior commissure to the vocal process of the arytenoid. Above and below, its borders approximate the transition point between stratified and cylindrical epithelium.

Hajek was able to inject this space with a stained mass.[2] In demonstrating its outlines he proved its outer border to be the elastic membrane or vocal ligament. The medial or inner border of Reinke's space is formed by stratified squamous epithelium extending 1.5 to 2 mm. in either direction from the free edge of the vocal cord. Garten states that 10 to 20 layers of stratified squamous epithelium are present except at the edge of the vocal cord and on its adjacent upper surface.[7] In the latter regions the epithelium is thinner and consists of only two or three layers. The thin epithelium forms a *locus minoris resistantiae,* which may give way before an increased pressure due to congestion and subsequent edema of Reinke's space. The elastic membrane, interspersed with muscle fibres laterally, prohibits extension of the edema in this direction. Above and below, the *lamina propria* varies somewhat in that it contains many mucous glands surrounded by elastic fibres. Here too, there exists a mechanism limiting the possible spread of any subepithelial edema.

The arteries of the vocal cord are scarce, but veins are numerous and large, forming three distinct superimposed nets. The most superficial venous network consists of elongated meshes. As a result of increased and prolonged pressure, thin-walled veins may easily become compressed and passive congestion may develop.

Thus it may be seen that anatomical factors predispose to the development of edema in the subepithelial space described by Reinke.

PATHOLOGY

In a careful study of a series of vocal cord polyps, the dominant microscopic features have been found by us to be congestion, edema, thrombosis and hyalinization.

Congestion is obvious in all sections at our disposal in the form of thin-walled vessels filled with erythrocytes.

The edema is at times so extensive that only a small amount of actual tissue remains within the polyp (Fig. 1). Some polyps remain edematous. An uncomplicated picture of passive hyperemia is only rarely observed, however, because of the degenerative alter-

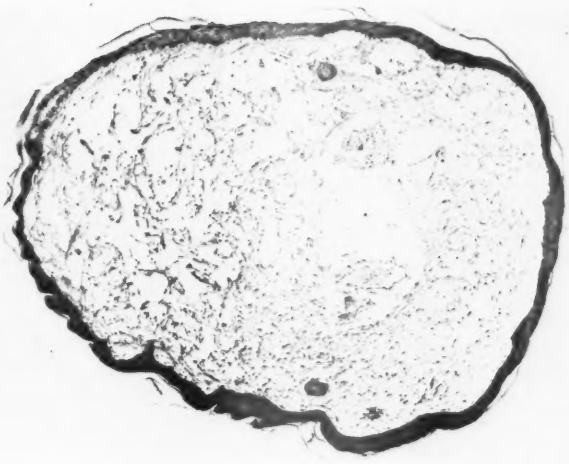

Fig. 1. Simple laryngeal polyp. Note the extensive edema, the dilated vessels, the sparsity of connective tissue elements and the lack of inflammatory reaction. (X80.)

ations which follow decreased circulation and impaired cellular nutrition. It is not known why such changes appear in some polyps and are absent in others.

Circulatory disturbances become evident in the development of thrombi. One may observe progressive stages in this development in Fig. 2. Whereas the thrombus in this illustration only partially fills a vessel lumen, almost complete thrombotic occlusion may be readily observed in Fig. 3.

In addition to thrombosis, more or less extensive hyalinization may be noted. This physical change apparently takes place shortly after thrombosis, a fact borne out by re-examination of Fig 2, wherein may be seen a young thrombus already partially hyalinized. Furthermore, hyalinization may involve the vessel walls and the sparse connective tissue elements of the polyp. It will be recalled that New and Erich also described amyloid changes in this connection.[1] (Differential staining was employed to certify the hyalin character of the degenerative products. Elastic and reticulum stains were made but disclosed only a sparsity of these tissues.)

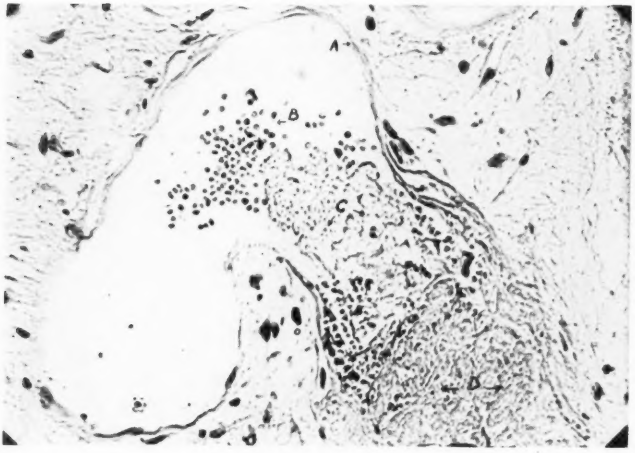

Fig. 2. Progressive stages in thrombus development within a vessel. At *A* the vessel wall; *B*, erythrocytes; *C*, fibrin deposition; *D*, extensive hyalinization with blending of the hyalin into surrounding connective tissue.

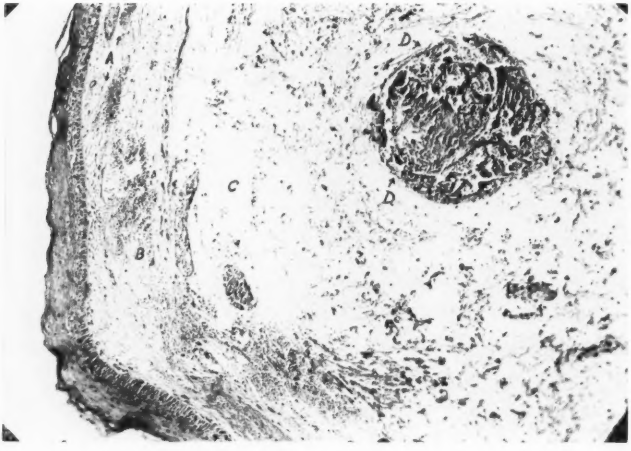

Fig. 3. Laryngeal polyp with thrombotic occlusion of large vessel. Beneath the epithelium may be seen: *A*, engorged vessels; *B*, erythrocytic extravasations; *C*, extensive edema; and *D*, hyalin thrombosis of large vessel.

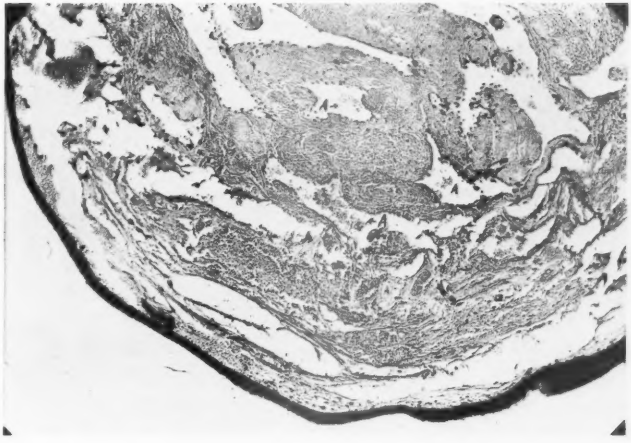

Fig. 4. Marked hyalinization of laryngeal polyp. Vessels containing blood elements may be seen at *A*. The hyalinization involves all of the stroma between the elongated tortuous vessels.

The aforementioned pathological features serve to explain clinical variations which occur. Some laryngeal polyps are pale and collapse on the slightest manipulation, whereas others are more firm in consistency and require that care be taken in order to remove them in their entirety. Figure 1 illustrates a polyp of the first group, in which edema is the principal characteristic. This contrasts markedly with Fig. 4, which shows an extensively hyalinized polyp.

The squamous epithelial covering of these polyps may vary in thickness. Although the usual number of cell layers may be present, other examples of hyperplasia and hyperkeratosis may be observed. It is conceivable that the epithelial thickening results from constant irritation. The germinal layer of the epithelium may occasionally show slight degenerative changes.

COMMENT

It is well known clinically that vocal cord polyps develop in individuals who are guilty of vocal abuse or misuse. It is also generally recognized that polyps, as well as singers' nodes, are usually seen at the junction of the anterior and middle third of the vocal

cord. Just why this is true may be explained by the foregoing anatomical concepts together with a consideration of vocal function.

Gutzmann has demonstrated stroboscopically the existence of some sort of "vibration node" at the point where polyps develop. Such points of the cord margins are at rest while bordering regions are in motion. It is thought that the cords do not come in contact at these particular "vibration nodes" and that actual concavities of the cord margin may exist there.

One might reason from these facts that stasis and the accumulation of blood could be expected at points of relative rest and concavity along the cord margin. The stasis and its consequences (edema, thrombosis and hyalinization) become more pronounced with increasing vocal strain. The tonus of the vocal ligament and the bordering vocal muscle may contribute to the maintenance of stasis.

It is also important to call attention to the acute polyps which suddenly develop following severe vocal strain. Their location is identical, and they are sometimes due to hemorrhage in Reinke's space. Pre-existing venous dilatations in this area may be predisposing factors.

It may be concluded that local anatomical factors together with functional disorders facilitate the development of vocal polyps. Changes of inflammatory nature, if present, are secondary and disappear following removal of the growth and elimination of functional disturbances.

SUMMARY AND CONCLUSIONS

1. Polypoid growths of the vocal cord margin are common and non-neoplastic in character.

2. A potential loose subepithelial tissue, known as Reinke's space, is the starting point in the development of these polyps.

3. Congestion, edema, thrombosis and hyalinization may be observed in the present series of polyps removed from vocal cord margins.

4. Physiological factors are also significant in the growth of these polyps.

5. Inflammatory reactions, if present, are secondary in nature.

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LVIII

ROENTGENOLOGY OF THE NECK AND MEDIASTINUM*

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CINCINNATI

In this paper some phases of the roentgen anatomy, physiology, and pathology of the neck will be considered. Fundamental X-ray studies of the anatomy and physiology were made some decades ago, but it is only during recent years that due consideration has been given to the clinical pathology revealed by the X-rays.

ANATOMY

Ossification of the Larynx. The roentgen rays are particularly valuable for the study of the normal process of ossification of the laryngeal cartilages, and in order to appreciate some of the pathologic conditions of the larynx it is important that one should be familiar with the usual course of ossification. According to the pioneer studies of Scheier[1] and Fraenkel,[2] ossification usually begins during the eighteenth or nineteenth year of life, and in both sexes is first noted in the lower part of the posterior border of the thyroid cartilages. From this region it gradually ascends along the posterior border. Ossification then gradually extends forward along the inferior border of the thyroid, giving rise to an L-shaped mass of bone.

In the male, a new center arises at the inferior thyroid notch and another along the superior thyroid border, and by the gradual coalescence of all these centers the cartilage eventually may be completely surrounded by a bony frame (after the fortieth year).

In the female the same process may occasionally occur, but more characteristic for this sex is the freedom from ossification in the anterior portions of the thyroid wings.

In the cricoid the process begins in the posterior lamina and gradually extends forward into the anterior arch of the cartilage.

The arytenoids are next in order, and the tracheal rings are among the last to show signs of ossification. (See Figs. 1 and 2.)

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Although the process of ossification usually takes place in the manner just recorded, recent studies by Chamberlain and Young[3] and by Taylor[4] indicate that in exceptional instances, especially in the presence of inflammatory lesions, the process may be accelerated (Fig. 2), while in other instances it may be retarded. From a practical standpoint it is important not to mistake a center of ossification for a foreign body. Ossification of the thyroid cartilage or the trachea may also interfere with the operations of thyrotomy or tracheotomy, as the case may be.

The Normal Neck of the Adult in the Lateral Roentgenogram. The cervical spine with its coverings forms the background of the image. In the lateral view the upper air and food passages are well delineated owing to the contrast between the translucent air column and the surrounding hard and soft parts. From above downward the following structures are revealed (Fig. 1): the root of the tongue, the valleculae, the hyoid bone with its greater cornua, and, projecting between these, the epiglottis. The posterior wall of the pharynx, hugging the bodies of the vertebrae, stands out sharply. The thyroid cartilage, more or less ossified, is very prominent. Next in order is the cricoid cartilage with the cricothyroid space. The conical-shaped arytenoids, and frequently the aryepiglottic folds, are seen posteriorly with the opening into the hypopharynx behind the arytenoids. The lumen of the larynx appears as a distinctly lighter area continuous below with the well-defined lumen of the trachea in which the rings are visible only when ossified. Under favorable conditions the true and false cords, as well as the ventricle of the larynx, are distinctly outlined. The posttracheal shadow represents the cervical esophagus with its anterior and posterior walls in contact, so that the esophagus outline is approximately twice as thick as that of the single-walled pharynx.

Lateral Roentgenogram of the Infant. The lateral roentgenogram of the infant presents a somewhat different picture from that of the adult. The neck is relatively shorter, and the vertebrae are only partially ossified. The hyoid bone and epiglottis are visualized, but the remaining cartilages are invisible. The pharynx, larynx and trachea are, therefore, outlined by the air column within them which stands out in sharp contrast with the surrounding soft structures. As pointed out by Hay,[5] who made accurate measurements, the pharyngeal wall is relatively much thicker in the infant than in older children, and the wall becomes distorted when the infant cries. It is, therefore, important that the roentgenogram be made while the infant is quiet. Pancoast and Pendergrass[6] have pointed out that

Fig. 1. Fig. 2.

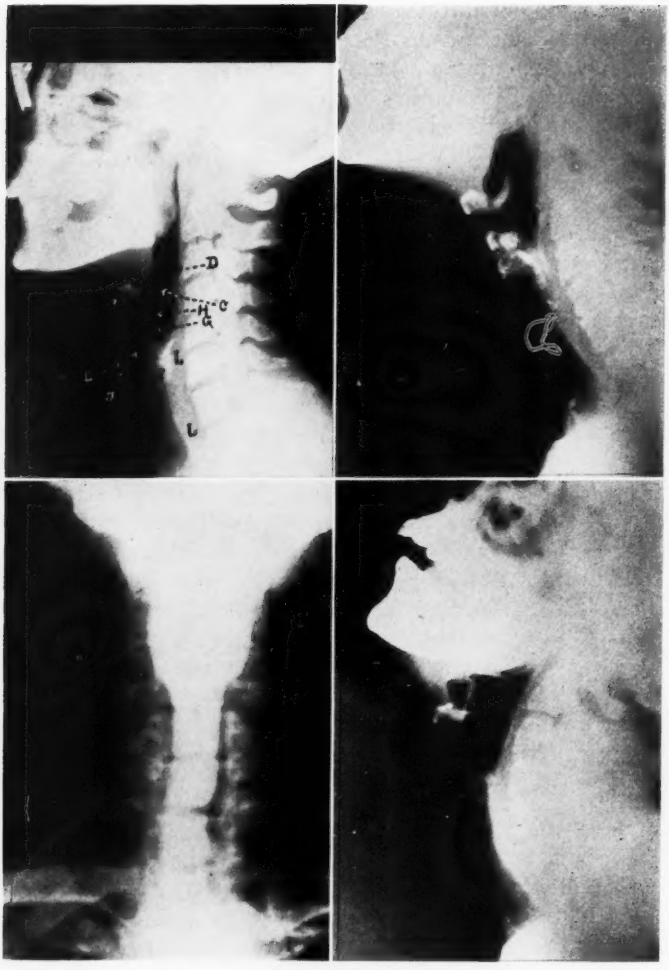


Fig. 3. Fig. 4.

Fig. 1. Female, aged 42. A—Root of tongue and valleculae; B—hyoid bone; C—epiglottis; D—posterior pharynx wall; E—thyroid cartilage; F—cricoid; G—arytenoids; H—ary. folds; J—ventricle; K—trachea; L—esophagus.

Fig. 2. Excessive ossification of laryngeal cartilages and tracheal rings attributed to X-ray treatment of a goitre. (Dr. S. Brown.)

Fig. 3. Anterior view. Barium-filled pharynx and esophagus. The pharynx is wide, the esophagus narrow.

Fig. 4. Patient singing "E-e-e". Note elevated epiglottis and open ventricle of Morgagni.

the best radiograms of the infant's pharynx and air passages are obtained during the inspiratory phase of respiration when the soft parts are expanded to admit the incoming air.

PHYSIOLOGY

Deglutition. Scheier[7] in 1911 was among the first to make roentgen studies of the act of deglutition and was able to portray the position of the bismuth mass in the fractional part of a second required for it to pass the larynx. More recent studies have been made by Mosher (1927),[8] Brown and Reineke (1928),[9] Barclay (1930),[10] and Pancoast (1933).[11] These authors employed both the fluoroscope and roentgenograms.

The various phases of the mechanism of deglutition occur in the following order: the opaque medium (bolus) is pushed upward and backward against the hard and soft palate by the action of the tongue while the soft palate becomes elevated and approximated to the posterior and lateral pharynx wall, closing off the nasopharynx. At this instant the base of the tongue tilts backward and comes in contact with the posterior pharyngeal wall. At the same time the hyoid bone, together with the larynx, is drawn upward under the base of the tongue, thereby bringing the arytenoids into contact with the epiglottis, which is supported by the base of the tongue. These combined maneuvers close off the vestibule of the larynx and obliterate the pharynx. In the next phase the lower pharynx, which has remained momentarily closed, suddenly opens, and the bolus is pushed over the epiglottis into the pharynx by a sort of dump-cart action of the tongue.

According to Barclay,[12] the sudden opening of the pharynx creates a partial vacuum which sucks the food into the pharynx and esophagus. Pancoast,[13] on the other hand, believes that the "receptive" pharynx takes hold of the bolus and "squeezes" it down into the upper esophagus, which takes care of it by peristaltic action aided more or less by gravity. Once in the esophagus, the opaque medium is propelled into the stomach with a slight delay at the cardia.

Deglutition should be observed both in the lateral and the antero-posterior positions (Fig. 3). Obstructions of any kind along the pathway taken by the bolus interfere with its passage and, under the fluoroscope, are manifested by delay, diversion, or stoppage of the opaque medium. Not infrequently under these conditions a spill-

over into the larynx and trachea can be observed. With a partially occluding foreign body in the hypopharynx or esophagus, the barium can sometimes be seen to pass about the foreign body like a stream flowing past an island. Occasionally peristaltic (and antiperistaltic) waves in the lower esophagus are pictured in the roentgenogram.

Phonation. The various positions assumed by the lips, tongue, soft palate, and larynx during the act of phonation were accurately determined in the pioneer roentgenography of Scheier in 1909.[11] In more recent years the physiology of the upper respiratory tract has been studied by Brown, McCarthy and Reineke[15] and others. Both fluoroscopy and roentgenography have been employed in these latter studies. The relative positions assumed by the larynx and the soft resonating structure above the larynx are best observed when the subject under investigation produces one of the vowel sounds. The particular changes in the position of the hyoid bone, the larynx, and the epiglottis varies with each vowel and is accompanied by a corresponding conformation or adaptation of the pharynx and soft palate, etc. During phonation, for example, the epiglottis attains its greatest elevation corresponding to clinical experience gained from mirror laryngoscopy (Fig. 4).

PATHOLOGICAL CONDITIONS

In the X-ray study of abnormal conditions in the neck, both fluoroscopy and roentgenography should be employed, the former to disclose faulty functioning of the parts, the latter for permanent record and as a guide to surgical procedure. Whenever needed, contrast media should be used to aid in better visualization of the pathological condition.

Foreign Bodies. The value of roentgenography in the detection of opaque foreign bodies is so well recognized that this subject will not be considered at any length in this paper. For the detection of non-opaque foreign bodies in the esophagus barium meal is usually employed, since the barium will usually adhere to the offending body. As I have pointed out in a former publication,[16] the use of lipiodol is sometimes to be preferred as a contrast medium since it does not, like barium, leave a deep tenacious pool above the foreign body, which may interfere with visualization through the esophagoscope. In high obstructions of the esophagus, as well as in cases of suspected tracheo-esophageal fistula, it is also better to employ iodized oil, since less irritation will ensue should the contrast medium pass into the

tracheobronchial tree. The error of confusing the ossified plaques in the laryngeal cartilages with foreign bodies has already been mentioned. This error can usually be avoided by a knowledge of the usual centers of ossification, as well as by the ingestion of a contrast substance. Figure 5 presents a good example of the difficulty in the visualization of a sliver of bone lodged behind the cricoid cartilage.

Tumors. Tumors at the root of the tongue and in the nasopharynx or hypopharynx can be demonstrated in the roentgenogram. Benign tumors usually have a smooth outline, whereas malignant growths are more irregular and invasive and usually interfere more with function. In advanced carcinoma of the larynx an X-ray picture may show more extensive involvement of the cartilage and surrounding tissues than is apparent at laryngoscopy and may be the deciding factor in determining the best type of operation. Occasionally the roentgenogram will show the presence of an unsuspected tumor in the trachea or in the party wall between the trachea and esophagus. I have encountered two such cases, one of which presented symptoms referable to both esophagus and trachea. A benign tumor within the larynx can also be visualized. When the tumor is pedunculated and attached to a vocal cord, it may be necessary to instruct the patient to phonate during the filming in order to bring the neoplasm into view.

STENOSIS OF THE LARYNX

Stenosis of the larynx and upper trachea may occur in both the acute and the chronic form.

Acute Stenosis. Hay,[5] Pancoast and Pendergrass,[17] and others have succeeded in demonstrating the narrowed air passages in infants and young children suffering from acute diphtheria, streptococcic laryngitis, and the like. The filming should be done in both the inspiratory and expiratory phases of respiration; the stenosis usually becomes more apparent in the inspiratory phase.

Chronic Stenosis. Chronic cicatricial stenosis is usually demonstrable in both children and adults.[18] In such conditions the degree of obstruction is visualized by the narrowing or distortion of the air column outlined against the denser surrounding parts (Fig. 6). It is sometimes difficult, however, to determine the upper and lower limits of the cicatrix. Under this condition in the tracheotomized patient the procedure illustrated in Fig. 7 may be employed. In this maneuver two sounds have been introduced, one above the other below the complete obstruction, thus demonstrating its vertical ex-

tent. In the treatment of cicatricial stenosis by the insertion of permanent dilators, it is advisable to determine by X-ray whether or not they are seated in proper alignment. Figure 8 shows a dilating, indwelling, folded, rubber tube employed in the treatment of a case of laryngeal stenosis. The film demonstrates that the tubing is in proper alignment.

For the instillation of lipiodol, especially in children, I prefer the use of the special self-retaining intubation tube which I devised some years ago. After the insertion of the tube, the patient may be placed in any desired position, and the direction of the flow of the lipiodol into any portion of the tracheobronchial tree can be readily controlled under the fluoroscope. Since the removal of the tube will excite a cough reflex, it is allowed to remain *in situ* until the roentgenogram has been completed. Figure 9 shows the tube in place, as well as the lipiodol which has entered the trachea and bronchi.

For teaching purposes it is sometimes advantageous to demonstrate the position of instruments introduced into the larynx, trachea, or esophagus. Fig. 10 shows a direct laryngoscope in proper position behind the epiglottis, hyoid bone, and thyroid cartilage, as well as demonstrating the correct position of the cervical spine.

COMPRESSION OF THE TRACHEA AND ESOPHAGUS

The trachea and esophagus may be compressed or partially occluded by the encroachment of adjacent masses, such as tumors or inflammatory exudates. Firm goitres may compress the trachea from side to side, producing the so-called scabbard deformity. This is best demonstrated in the anteroposterior roentgenogram.

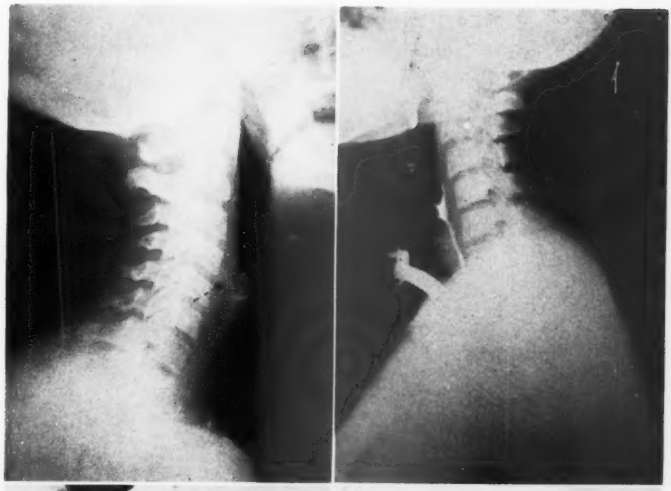
In compression by the isthmus of the thyroid gland, a lateral view will demonstrate the lesion. The carcinomatous goitre usually gives a dense shadow, and fluoroscopy may show impaired or absent motion of the arytenoid cartilage if the articulation or recurrent laryngeal nerve is involved.

Swelling and edema of the epiglottis and arytenoid folds (tuberculosis) can be readily visualized in the X-ray. When the patient cannot open his mouth, sometimes this method of diagnosis yields valuable information.

EXOSTOSIS OF THE CERVICAL SPINE

Mosher[8] has called attention to cervical exostoses as a cause of difficulty in swallowing. Such exostoses are, of course, apparent in

Fig. 5. Fig. 6.



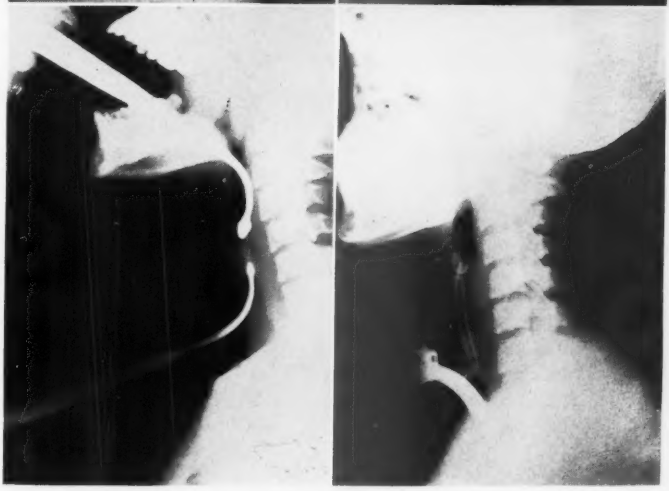


Fig. 7. Fig. 8.

Fig. 5. Arrow points to a sliver of bone in the esophagus, lodged behind the partially ossified cartilages of the larynx in a woman, aged 29.

Fig. 6. Showing stenosis of the larynx and trachea above the tracheal cannula. (Arrows) Barium in the esophagus.

Fig. 7. Girl, aged 15 years. Complete stenosis of larynx. Showing method of measuring vertical extent of the obstruction. Note calcification of larynx.

Fig. 8. Showing folded dilating rubber tubing in a stenosed larynx.

Fig. 9. Fig. 10.

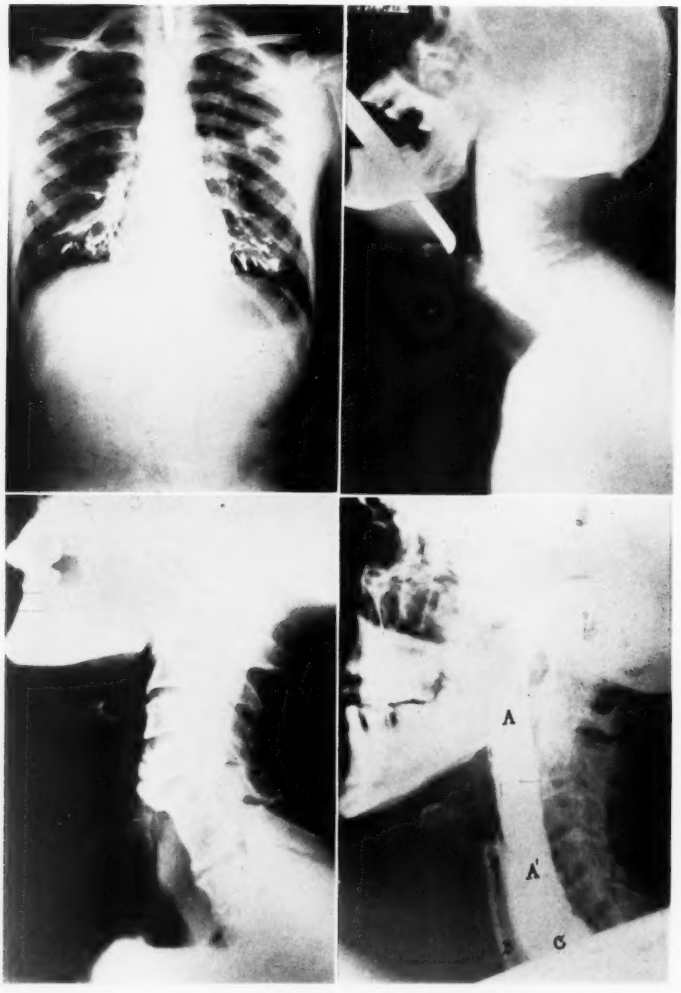


Fig. 11. Fig. 12.

Fig. 9. Boy, aged 12 years. Showing intubation tube through which lipiodol has been introduced. Bronchiectasis.

Fig. 10. Direct laryngoscopy. Showing correct alignment in the larynx and proper position of the spine.

Fig. 11. Showing osteochondroma arising from the bodies of the fifth and sixth vertebrae, encroaching upon the esophagus. The bodies of the sixth and seventh vertebrae are fused.

Fig. 12. Cadaver of an adult. Roentgenogram showing the retrovisceral space filled with radiopaque gauze (A-A') introduced from below. (B) a metal pipe in the esophagus. It will be noted that the "retropharyngeal" space (A) is contiguous with the retro-esophageal space (A') and the posterior mediastinum (C).

the radiogram, and the degree of obstruction can be determined by the barium meal. In 1938 I reported a typical case,[19] as demonstrated in Fig. 11, which shows an osteochondroma of the cervical spine which caused marked interference with swallowing. Under the fluoroscope the opaque medium was seen to enter the right pyriform sinus, where it failed to pass but detoured into the esophagus through the left pyriform sinus. The osteochondroma was removed through an anterior cervical approach, with prompt recovery of the patient. This, so far as I am aware, was the first case in which a cervical exostosis was removed for the relief of dysphagia. The patient reports that he has had some difficulty in swallowing solids during the past three months, but there is no recurrence of the osteochondroma. The present trouble seems to be due to some neuromuscular disturbance.

RETROPHARYNGEAL AND RETRO-ESOPHAGEAL INFECTIONS, AND MEDIASTINITIS

There are five potential connective tissue spaces about the pharynx. On each side, the parapharyngeal, or pharyngomaxillary, space lies lateral to the pharynx. Behind the pharynx musculature there is a closed right and left intramural peripharyngeal space (Weintraub[20]). Posterior to the peripharyngeal spaces there is a single wide extensive retropharyngeal space continuous below with the retro-esophageal space, the two together constituting the retrovisceral, or postvisceral, space which terminates below at the diaphragm.

All of the spaces contain loose areolar spiderweb-like connective tissue permitting free movement of the pharynx and esophagus. This elastic areolar tissue, when infected and inflamed, may become greatly distended and will then encroach upon or dislocate adjacent structures. This distention is well illustrated in Fig. 12, showing a radiogram of the neck of a cadaver in which radiopaque gauze has been packed into the retrovisceral space from below without any previous dissection of the parts. In a similar manner in interstitial emphysema of the neck from any cause, the retrovisceral space may become greatly distended with air, as demonstrated in Fig. 13.

Acute infection in any of the neck spaces occurs in three forms, namely: as a lymphadenitis, as an abscess, or as a cellulitis (phlegmon). Abscess formation or cellulitis may follow a lymphadenitis, a severe pharyngitis, or trauma to the pharynx or esophagus with or

Fig. 13. Fig. 14.

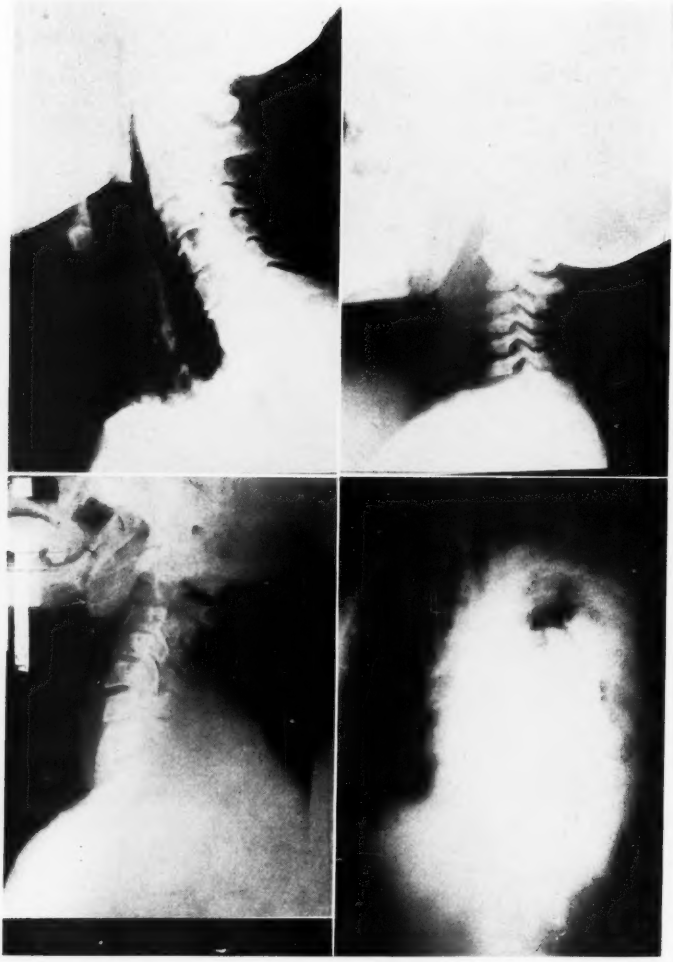


Fig. 15. Fig. 16.

Fig. 13. Interstitial emphysema of the neck with marked distention of the postvisceral space.

Fig. 14. Large retropharyngeal abscess in a child, aged 4 years.

Fig. 15. Young man. Arrows point to a "cold" abscess originating from carious first and second vertebrae.

Fig. 16. Ruptured esophagus with free barium in the pleural cavity and air in the tissues.

without perforation of the muscular coats of these organs. Furthermore, an abscess in adjacent spaces or structures may gravitate into the postvisceral space which, according to Pearse,[21] constitutes the pathway of descending cervical infections in 71% of all cases. In the diagnosis and localization of these lesions, roentgenograms are of exceeding importance and give invaluable information.

Retropharyngeal (Peripharyngeal) Abscess. In the case of a suspected retropharyngeal abscess in an infant, it is advisable, whenever possible, to take a lateral radiogram of the neck before the child has been disturbed by other methods of examination. The best pictures are obtained during the inspiratory phase of respiration when the pharynx is distended. Figure 14 illustrates such a case with marked convex forward protrusion of the posterior pharyngeal wall encroaching upon the lumen of the pharynx and to some extent upon the vestibule of the larynx. From this roentgenogram it is apparent that such abscesses can readily be opened and drained through the mouth provided precautions are taken to prevent the aspiration of pus.

Cold Abscess. A cold abscess, originating from caries of the body of one or more of the upper cervical vertebrae, may cause a swelling behind the pharynx and may at times be difficult to differentiate from the ordinary acute abscess. There is usually a history of more gradual onset accompanied by rigidity and pain in the neck. In the differential diagnosis the roentgenogram will show the retropharyngeal swelling, together with signs of erosion, in the affected vertebrae (Fig. 15). The differentiation is important because the cold abscess should be approached by an external route in order to avoid contamination from the buccal flora.

Retro-esophageal Infection. Infections behind the pharynx and the cervical esophagus cause a forward dislocation of the pharynx wall, as well as of the esophagus and trachea, and present quite characteristic pictures in the lateral view. Anteroposterior roentgenograms should also be taken and may reveal a widening of the superior mediastinal shadows. Figure 17 shows enormous inflammatory distention of the retropharyngeal and retro-esophageal spaces with forward dislocation of the trachea. Figure 18 gives the anteroposterior view. It is apparent that such cases require an external approach for drainage. In this case at operation the distention was found to be due to a diffuse cellulitis. The patient recovered after a stormy convalescence during which pictures taken at intervals showed a gradual decrease in the swelling.

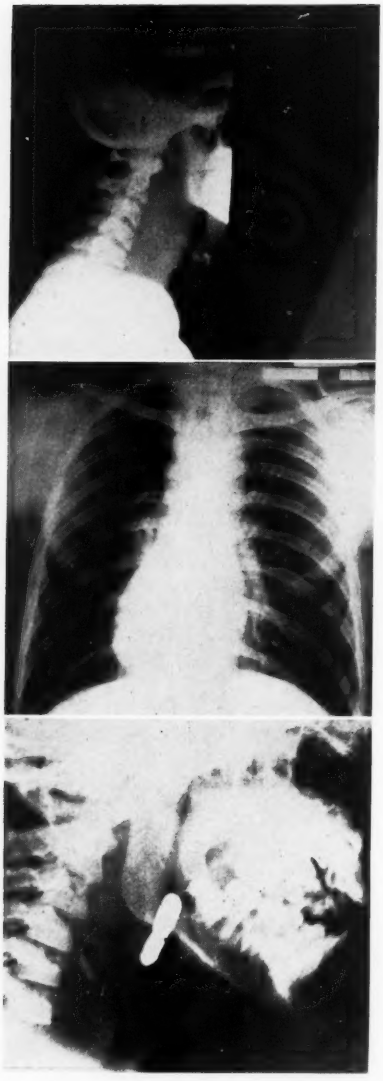


Fig. 17. Cellulitis causing massive retropharyngeal and retro-esophageal swelling extending into the posterior mediastinum.

Fig. 18. Anteroposterior view of same patient as Fig. 16 showing widening of mediastinal shadow.

Fig. 19. Foreign body which had perforated the esophagus seen lying free in an air-containing abscess cavity. (The bismuth capsule is arrested above the constriction.)

PERFORATION OF THE ESOPHAGUS

There are two cardinal X-ray signs of perforation, or rupture, of the esophagus. The first sign, interstitial emphysema, arises from the escape of air into the periesophageal connective tissues where it becomes visible in the radiogram. The second sign may occur if a radiopaque meal has been administered and some of the opaque substance appears external to the esophageal lumen. The air which escapes from the esophagus may find its way to the root of the neck where it can be palpated. In certain instances, however, the air may become pocketed near the perforation and may not appear in the neck at all.

In perforation of the hypopharynx and upper esophagus the lateral roentgenogram will reveal both the periesophageal swelling and the air or gas bubbles in the connective tissues. This is important since early diagnosis and prompt surgical intervention are essential in this condition. Figure 19 shows a foreign body external to the esophagus with an air bubble about it. This was the first roentgenogram of the kind reported in this country[22] and was not correctly interpreted until esophagoscopy had exposed the site of perforation with the absence of the foreign body. After the correct interpretation of the roentgenogram, the foreign body was removed and the abscess drained through an external incision. The patient recovered.

Figure 16 is the roentgenogram of a patient who was admitted to the surgical service in profound shock and with marked abdominal symptoms. A barium meal was administered, and the stomach and left chest were seen to contain a large amount of the barium. The X-ray findings were thought to represent a herniation of the stomach through the diaphragm. A laparotomy was performed, but the patient died. At autopsy an ulcer was found in the terminal portion of the esophagus with rupture of the esophagus at that point, together with free barium in the left pleural cavity. In the light of the information gained from the autopsy it became apparent that a correct interpretation of the film might have been made since both air bubbles and barium were visible in the left chest region. The large quantity of barium visible in the chest had been very misleading.

* * *

In this paper no mention has been made of X-ray therapy for malignant disease of the larynx and pharynx, which occasionally yields such phenomenal results. The subject of laminography has

also been omitted. From all reports this procedure promises to widen further the field of the roentgen study of the neck.

SUMMARY

1. The roentgen anatomy of the neck is described with particular emphasis on the ossification of the laryngeal cartilages and the roentgenogram of the normal neck.

2. The roentgen physiology of deglutition and of phonation is briefly described.

3. The X-ray findings in various pathological conditions, including tumors, laryngeal stenosis, spinal exostoses, retropharyngeal and retro-esophageal infections, mediastinitis, and perforation of the esophagus are discussed.

I wish to express my appreciation to Dr. Samuel Brown and Dr. Archie Fine for their assistance and cooperation in the preparation of this paper, and to Mr. Ralph Simcox for his care in producing the illustrations.

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LIX

CAROTID SINUS SYNDROME

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AND

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During recent years a new clinical entity has been observed and studied, known as carotid sinus syndrome. Certain characteristic symptoms of this syndrome are frequently found in certain otological complications. It was felt by the authors that a brief review of carotid sinus syndrome was indicated, along with a case history of a patient who presented such a syndrome and was successfully cured by operation.

ANATOMY

The carotid sinus is a bulbous dilatation at the bifurcation of the common carotid artery, situated in the neck at the level of the superior border of the thyroid cartilage or slightly above it. This dilatation involves mostly the origin of the internal carotid artery. It is richly supplied, particularly in the adventitia, by sensory receptor nerves which terminate in characteristic menisci. These nerves emerge to constitute the sinus nerve of Hering, formerly known as the *ramus caroticus hypoglossi.* Hering's nerve is found between the two branches of bifurcation of the common carotid artery; it then ascends to join the glossopharyngeal nerve. According to Wright[1] the wall of the carotid sinus is thinner than that of the adjacent part of the artery, due to a decrease in the muscle fibers of the media.

The anatomy of the carotid sinus hase been particularly well studied by Hovelacque, Maes, Binet and Gayet,[2] Ask-Upmark,[3] Ferris, Capps and Weiss,[4] Danielopolu[5] and Tchibukmacher.[6]

The carotid sinus has numerous anastomotic branches with surrounding nerves. Through the sinus nerve of Hering it is connected with the glossopharyngeal nerve. It has connections with the vagus, the upper pharyngeal branches of the vagus, the superior cervical ganglion of the sympathetic. There is also an anastomosis with the carotid body.

The carotid body, a small glandular mass lodged in the bifurcation of the common carotid artery, has a role that has not been clarified. It is constituted of glandular cells, arranged in clumps, and presents a rich vascular supply.

PHYSIOLOGY

Our present knowledge of the physiology of the carotid sinus is largely due to the pioneer work done by Hering[7] and Heymans.[8]

The carotid sinus represents one of the most important regulatory mechanisms of the blood pressure. By its reflex action upon the parasympathetic and sympathetic systems, upon the adrenal glands and probably other important structures, it tends to prevent too dramatic variations in the blood pressure. Elevation of the intranasal pressure produces instantaneous cardiac inhibition, vasodilatation and a drop in the blood pressure, whereas a fall of the intranasal pressure produces a cardiac acceleration, vasoconstriction, emptying of the spleen and adrenal secretion.

This "buffer" action is also presented by the aortic depressor nerves, and a similar mechanism exists, according to Heymans,[8] in the splanchnic arteries, in the superior vena cava and in the first portion of the pulmonary arteries.

Hering[7] showed that afferent impulses were carried from the carotid sinus through the sinus nerve of Hering and the glossopharyngeal nerve (also in part through the vagus, the sympathetic and the hypoglossus), to the central nervous system and mainly to the medulla from where afferent impulses are transmitted.

The nerve endings in the carotid sinus are of two types:

1. Presso-receptors, situated in the sinus wall and sensitive to blood pressure changes;

2. Chemoreceptors, situated in the carotid body and sensitive to chemical stimuli such as lack of oxygen and increase in CO_2.

Placed as it is at the portal of entry to the vascular channels of the brain, the carotid sinus is important in regulating cerebral circulation. De Castro[9] mentions the fact that in cows the internal carotid artery is absent; instead there is a large occipital artery presenting sinus structures apparently possessing the same function as the carotid sinus in man.

A change in the cerebral circulation occurs as one element of the systemic change of the blood pressure. According to Forbes and

Cobb,[10] the cerebral vessels present both constrictor and dilator nerve fibers. These nerve fibers present only one-tenth of the activity of those of the skin. It appears probable that there is also a direct reflex action through the carotid sinus upon the cerebral vascular tone.

CLINICAL PATHOLOGY

There is evidence to indicate that under certain circumstances in man the carotid sinus may become unduly hyperactive and lead to syncope and similar manifestations.

Czermak[11] (1886) observed upon himself that pressure in a determined point of the right side of the neck produced a sensation of weakness with vertigo and slowing of the heart rate.

Hering[7] thought that the unconsciousness produced by the knock-out blow of boxers could be of carotid sinus origin. Ferraro[12] gave a similar opinion.

Roskam[13] (1930) gave a complete case report in which attacks of syncope were brought about by pressure over the carotid sinus. However, the most complete study of the clinical pathology of carotid sinus syndrome has been made by Soma Weiss and his co-workers.[4]

The evidence that hyperactivity of the carotid sinus is responsible for syncopal attacks is based upon the history and character of these attacks occurring following mechanical stimulation of the carotid sinus and upon the observation of attacks on numerous patients. Novocaine block of the carotid sinus prevents the attacks produced by pressure over the carotid sinus, and the denervation operation abolishes the attacks.

The symptoms of the carotid sinus syndrome are quite typical. The patient feels weak, then faint, has an intense sensation of dizziness, often a sensation of vacuum on that side of the head, then loses consciousness. In the milder forms syncope is not complete; the patient merely feels faint or dizzy.

These attacks can be induced experimentally in a patient with a hyperactive carotid sinus. The best procedure is to have the patient sitting on a chair and then gradually exert pressure along the anterior border of the sternomastoid muscle at the level of the superior border of the thyroid cartilage. The patient will feel faint, then slump forward into unconsciousness. Following the release of the pressure over the carotid sinus, consciousness will gradually return, usually after a period of a few seconds to a few minutes.

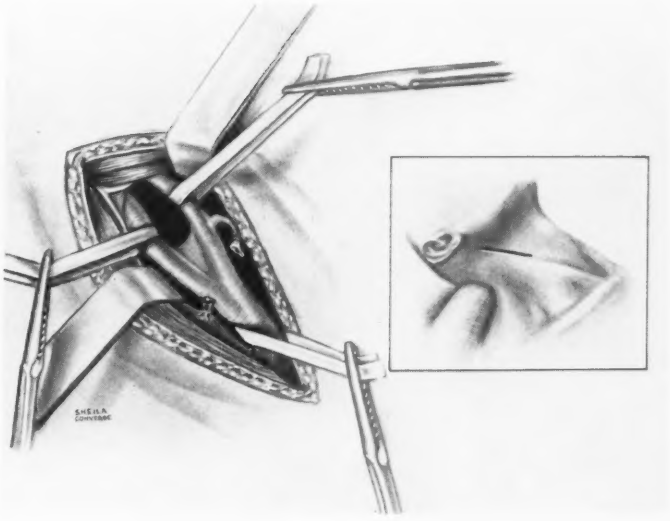

Fig. 1.

During such an attack certain changes occur in the blood pressure and the heart rate which can be recorded by means of a blood pressure apparatus and by taking the pulse rate. Upon these changes Soma Weiss has classified cases of carotid sinus syndrome into three groups:

1. The vagal type, in which the symptoms are due to a reflex slowing of the pulse rate through vagal inhibition of the heart and a fall in the blood pressure. Atropine by diminishing the sensitivity of the vagus, and adrenalin by increasing the irritability of the myocardium and raising the blood pressure, counteract and prevent the syncopal attacks.

2. The depressor type, which is less frequent. Here the syncope attacks are due to a direct reflex fall of the blood pressure without any change in the heart rate. It is due to the vasodepressor reflex of the carotid sinus producing a peripheral vasodilatation and a fall in blood pressure. Atropine has no effect. Adrenalin by producing peripheral vasoconstriction and raising the blood pressure diminishes the attacks.

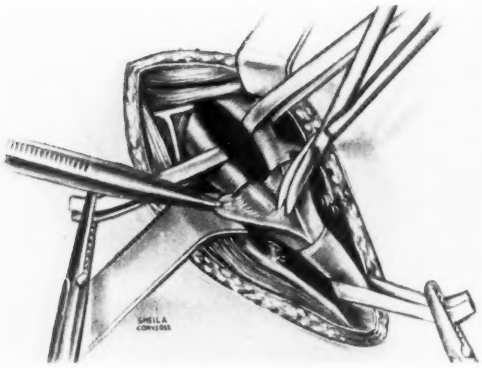

Fig. 2.

3. The cerebral type, characterized by the same attacks of syncope but without any changes in the pulse rate or the blood pressure. Neither atropine nor adrenalin have any effect in preventing the attacks. There has been a great deal of surmising concerning the cause of the syncope due to a hyperirritability of the carotid sinus. Claude Bernard attributed the loss of consciousness to cerebral ischemia. If this were so obliteration of the common carotid artery would cause unconsciousness, which this procedure does not.

This same argument may be applied where unconsciousness is attributed to a fall in the systemic blood pressure, thus producing a cerebral ischemia. It would not explain the syncope in the cerebral type of Soma Weiss, in which there is no drop in blood pressure. Forbes and Cobb[10] have shown that the cerebral vessels present both constrictor and dilator nerve fibers. Weiss and his co-workers obtained evidence that the syncope was associated with cerebral vasoconstriction followed by the vasodilatation, but the tendency to faint was not proportional to the vasomotor changes. They feel that the passage from the normal state to one of ischemia, producing a temporary anoxia, may be the cause of the syncope.

There may be some direct reflex action upon the centers of consciousness in the midbrain. These centers have been situated in the diencephalon, particularly in the optic thalamus,[11] in the inner

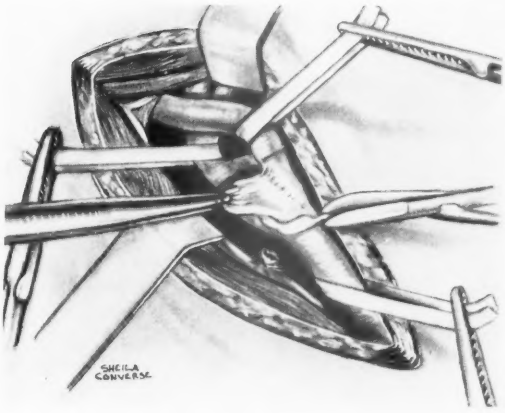

Fig. 3.

side of the caudate nucleus and septum pellucidum,[15] in the lateral hypothalamic region and in the supramammillary decussation,[16] in the diencephalon where the aqueduct of Sylvius opens into the third ventricle.[17]

ETIOLOGY

In some patients symptoms of carotid sinus syndrome appear without any apparent cause. In others they appear following an emotional upset or following mechanical stimulation by a tight collar, or because of overlying hypertrophied lymph glands or a tumor, an aneurysm of the carotid artery, a hypertrophic sternomastoid muscle. Certain drugs, such as digitalis, have a sensitizing effect. Their use preoperatively before a general anesthesia should be discouraged. A number of anesthetic deaths can be attributed to this practice, as shown by Weiss and by Rovenstine and Cullen.[18]

DIAGNOSIS

The possibility of a carotid sinus syndrome should be considered in the presence of a patient presenting unexplained attacks of syncope. The clinical diagnostic test is the reproduction of the symptoms, total loss of consciousness or faintness and dizziness, by pressure over the region of the carotid sinus.

An excellent method which will not only confirm the diagnosis but also demonstrate what result can be anticipated following the surgical denervation of the carotid sinus, is the novocaine block as performed by Wertheim and Rovenstine.[18] It is carried out upon the patient supine and head extended. The level of the transverse process of the fourth cervical vertebra is located. "The bifurcation of the common carotid artery is usually at this level and slightly anterior. After a skin wheal is raised at this point, a 50 x 7 mm. small gauge needle, unattached from the syringe is introduced. Because of the superficial location (1-3 cm.), caution is advised as the needle is advanced to contact the transverse process. After such contact the needle is withdrawn slightly, directed ventrally and advanced not more than 1 cm. Pulsation of the artery is felt and is transmitted through the needle. The syringe is then attached and with repeated aspirations and careful stabilization of the needle to prevent puncture of the large vessels, approximately 10 cc. of a 1% solution of procaine is deposited. Light massage at the site facilitates diffusion of the injected solution about the sinus area. Successful blocking of the area frequently is accompanied by partial recurrent laryngeal nerve paralysis which is manifested by hoarseness and by a subjective sensation of mucous in the trachea with a resultant cough."

TREATMENT

Conservative treatment should be instituted in the milder forms of carotid sinus syndrome. Rest in bed, the avoidance of emotional strain, the wearing of a loose collar to avoid compression of the sinus, suitable medication, atropine, adrenalin, neosynephrin, benzedrine in suitable doses and depending on the type of case, have been reported successful.

Where the attacks of syncope are frequent, alarming to the patient and to his family, and resistant to medical treatment, surgical denervation should be considered. A simple nervectomy of the carotid sinus nerve of Hering may be performed, but preferably a complete denervation of the carotid sinus should be done.

The operative procedure used is described and illustrated in the following case report.

REPORT OF A CASE

B. C., a white male, aged 42, of Irish extraction, was admitted to the Manhattan Eye, Ear and Throat Hospital, January 5, 1940, with the complaint of pain

in his right ear of 12 hours' duration. There was no antecedent history of acute upper respiratory infection or of previous aural pathology.

Physical examination at that time revealed the following positive findings: pain and tenderness over the right mastoid tip; M.T.A.D injected and thickened but devoid of bulging and without visible perforation; thin yellow discharge in right external canal; teeth dirty with moderate caries and early pyorrhea; old midline abdominal, right rectus and right subcostal scars; slight cyanosis of extremities with early clubbing of the fingers; blood pressure 118/80.

The past history was interesting only in the following respects: appendectomy in 1918 while with the A. E. F., followed shortly thereafter by exposure to gas with slow but complete recovery; operative intervention for duodenal ulcer in 1923, the nature of the operation being unknown; cholecystectomy and another operation for duodenal ulcer in 1927. Family and marital history were noncontributory.

An audiogram at this time showed the patient to have an average decibel loss of 47 db. for AD and 25 db. for AS while rotation studies were within normal limits. The right mastoid was reported as being sclerotic and non-cellular by X-ray. Laboratory findings were as follows: total WBC 7,600 with 60 polymorphonuclear leucocytes, 26 small lymphocytes and 14 transitional cells; 4.2 million RBC with 80% (D); Wassermann and Kahn tests negative: urinalysis negative; 2 plus benzidine reaction for occult blood in the stool.

The diagnosis of chronic mastoiditis right, with exacerbation was made, and on January 13, 1940, right radical mastoidectomy was carried out, the wound being packed open. Recovery proved uneventful, and on January 24, 1940, plastic closure with skin graft was done. The patient was discharged on February 7, 1940, as improved, there being at that time slight discharge from both the external canal and the postauricular wound.

On February 17, 1940, the patient was readmitted with the complaint of recurrent episodes of syncope. The first such attack had occurred the day following discharge and took place while he was lying in bed, there being no premonitory symptoms. Several days thereafter while waiting to have his mastoid wound redressed, the patient again fainted without apparent cause. The day before admission the last such episode occurred while the patient was riding on the subway. In each case the attack was not related to any postural change or change of head position, nor could any history of attacks of this nature in the past be elicited. The day on which the first attack of syncope occurred, the patient had noticed a small lump the size of a marble over the region of the right carotid sinus. This mass was non-tender, movable, firm and subsided almost completely after the repeated applications of cold compresses. Transient sore throat preceded this swelling, but by the time the swelling was noted such had completely disappeared.

Findings on physical examination were essentially the same as on the previous admission except that the postauricular wound was still draining moderately, and in addition pressure over the right carotid sinus was found to cause immediate syncope with a fall in blood pressure of 10 to 15 points (systolic) and marked slowing of the pulse. Over this carotid sinus region the pulsation of that vessel appeared unduly prominent, and in addition, a small non-tender, movable, pea-sized mass could be palpated. A tentative diagnosis of carotid sinus syndrome was

made and careful blood pressure and pulse rate studies carried out to confirm this diagnosis. Data from such studies were as follows:

B. P. Sitting up (at heart level): Rt. Arm 118/84; L. Arm 112/78.

Standing up (at heart level): Rt. Arm 124/86; L. Arm 120/78.

Lying down (at heart level): Rt. Arm 118/68; L. Arm 114/60.

Lying down: Pressure, left arm, 114/60. Right carotid sinus stimulated—fall to 90/70.

Sitting up: Pressure, left arm, 112/78—pulse 90. Right carotid sinus stimulated—fall to 84/64 and pulse to 60. Patient fell to recumbent position and became unconscious after first becoming pale with generalized sweating and after complaining of severe vertigo. No nystagmus occurred.

Stimulation of the left carotid sinus caused no change in blood pressure or pulse rate nor were untoward symptoms noted.

X-rays of the cervical spine were taken to rule out the possibility of cervical spine tumor, transverse process, and such were reported as being within normal limits. X-ray studies of the petrous pyramids revealed no pathology.

Since these data confirmed the diagnosis of carotid sinus syndrome, and since it was felt that the small mass which might conceivably represent a malignancy, could be the cause of the carotid sinus sensitivity, operative intervention was deemed advisable. Accordingly on March 8, 1940, a right pericarotid sinus sympathectomy was performed by one of us (Marvin F. Jones) in the following manner:

Operation.—A classical incision was made along the anterior border of the right sternomastoid muscle extending from the angle of the mandible to the level of the thyroid cartilage (Fig. 1, inset); the fascial planes were incised and the carotid sheath located; the carotid artery, internal jugular vein and vagus nerve were identified; the bifurcation of the common carotid was then located (Fig. 1); the carotid sinus appeared moderately dilated; the adventitia was carefully stripped for a distance of 2 cm. above the bifurcation and 1 cm. below by means of blunt scissor dissection (Fig. 2) and the dissection was carried around and behind the sinus, thus completely severing the carotid sinus from its nervous connections (Fig. 3). The wound was closed in two layers.

Two pea-sized lymph nodes reported by pathological examination to be hypertrophic, were found lying on the carotid sheath in the region of the carotid sinus. These represented the mass palpated on physical examination.

Following the operation the patient made an uneventful recovery and was discharged on March 18, 1940, symptom free. Since that time he has been seen periodically in the follow-up clinic. There have been no more attacks of syncope, the mastoid and cervical wounds have healed cleanly and otorrhea has ceased. In addition, the patient has gained considerable weight and states that subjectively he "never felt better in his life."

Stimulation of the right carotid sinus does not give rise to any of the previous train of symptoms or signs.

SUMMARY

1. A review of the anatomy, physiology and clinical pathology of the carotid sinus is outlined.

2. Methods of diagnosis and treatment of the carotid sinus syndrome are given.

3. The case history of a patient presenting a carotid sinus syndrome who was cured by surgical denervation is reported.

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LX

HISTOPATHOLOGICAL CHANGES OCCURRING IN CHRONIC INFECTION OF THE PHARYNX

HARRY P. SCHENCK, M.D.

PHILADELPHIA

The systemic effects of acute and chronic infection in the lymphoid structures which are the components of Waldeyer's ring concern every field of medicine, but the recognition and treatment of abnormality in these structures is the especial responsibility of the otolaryngologist. This responsibility is onerous in the presence of chronic infection and particularly when portions of the ring have been removed prior to the onset of the presenting symptoms.

Only minor differences of opinion persist as to the etiology and significance of acute infections in these lymphoid structures. Concerning the effects of systemic disease upon the pharyngeal lymphoid elements or the effects of chronic infection in the lymphoid tissue upon systemic disease, there are major differences of opinion.

It has long been recognized that the elimination of all infected lymphoid tissue in Waldeyer's ring cannot be accomplished by surgical measures alone. The disconcerting phenomenon of compensatory hypertrophy so commonly follows the removal of obviously diseased lymphoid tissue that repeated postoperative examinations become necessary. In addition, residual tissue, often septic and covered with mucous membrane or scar tissue, is found so frequently after surgical intervention that no deductions are possible as to the effect of tonsillectomy and adenoidectomy upon the incidence or morbidity of rheumatic fever, chorea, arthritis and similar diseases. Most internists are so thoroughly convinced of the finality of surgical removal of tonsils and adenoids that erroneous concepts have crept into the literature. Especially misleading are those reports attempting to establish the effects of such operations upon the incidence and course of systemic diseases while completely omitting all reference as to the amount of residual tissue, the degree of compensatory hypertrophy or the presence of infection in the residual portions of Waldeyer's ring. Until accurate and thorough studies are made in this direction, the concept of focal infection can be neither proven nor denied.

Fig. 1. Normal adult pharyngeal tissue. (X72.)

Clinical attention has been chiefly directed toward the pharyngeal, faucial, lingual and tubal tonsils with little emphasis upon the lymphoid follicles so generously distributed throughout the pharynx. Yet such follicles are an integral part of Waldeyer's ring and subject to the same influences and the same infections as the larger lymphoid masses.

The aggregations of lymph cells which are scattered throughout the pharynx are not only susceptible to acute and chronic infection, but they undergo alterations of cellular arrangement and structure in response to many systemic abnormalities. Thus in allergic children there occurs such marked alteration in the lymphoid structures of Waldeyer's ring that some allergists have attributed all hypertrophy of the tonsils and adenoids to an underlying allergy. The fallacy of such conclusion can be readily demonstrated by examining a group of non-allergic children. Moreover, if histological sections are studied, the reason for the apparent hypertrophy of the lymphoid

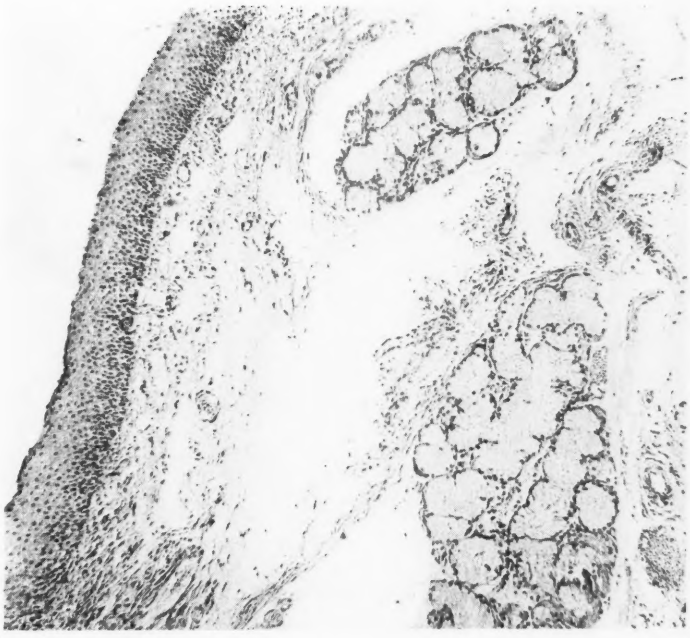

Fig. 2. Normal pharyngeal tissue from a child. (X306.)

elements in the allergic individual becomes evident. One finds that there is no actual increase in the number of lymphocytes contained in individual follicles, but that there is a striking increase in eosinophils and in polyblasts. The conspicuous size of the lymph follicle, however, is found to be due to edema, the profuse accumulation of intercellular fluid being facilitated by the loose structure of the follicle.

The pharyngeal lymph follicles also mirror certain changes in the body incident to other systemic diseases. Thus in leucemia, granulocytopenia, infectious mononucleosis, pernicious anemia, polycythemia and *purpura hemorrhagica* there occur specific alterations in the follicular structure which may be utilized for diagnostic purposes. In Hodgkin's disease, the follicles offer an accessible source of biopsy material.

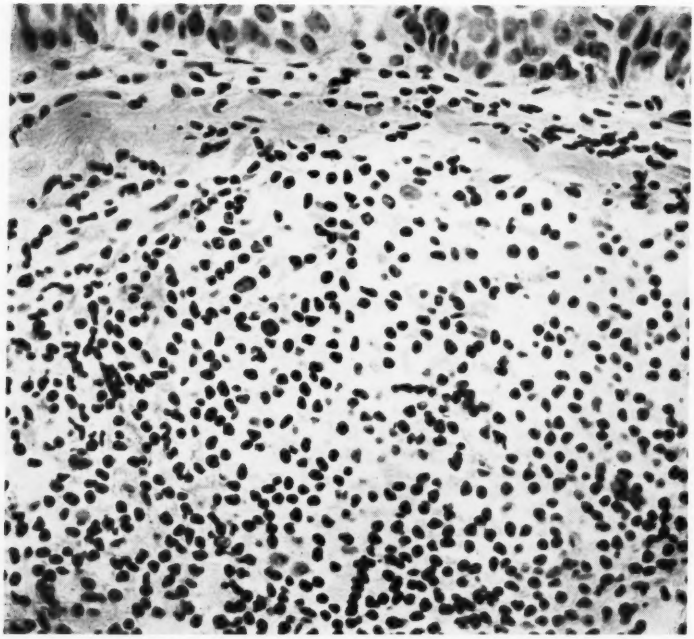

Fig. 3. Normal tissue from the same nodule shown in Fig. 1. (X306.)

A clear understanding of the pathology of the pharyngeal lymph follicles is essential to any discussion of chronic disease in the pharynx. Moreover, a clear distinction must be made between pathological alterations due to systemic disease and those resulting from primary involvement of the pharynx itself. When chronic disease of the pharynx is primary, the resulting pathologic changes are almost exclusively concerned with the lymph follicles. Any accompanying alterations in the mucosa, submucosa, glands or muscles are secondary to the follicular involvement. The gross and microscopic appearances of the pharynx in chronic infection depend entirely upon the duration of the disease, the identity of the invading micro-organisms and the immunological characteristics of the individual. Any division into separate groups or types of chronic pharyngitis is therefore purely arbitrary.

Chronic infection is not limited to those portions of the pharynx viewed in a cursory examination. The pharynx extends from the

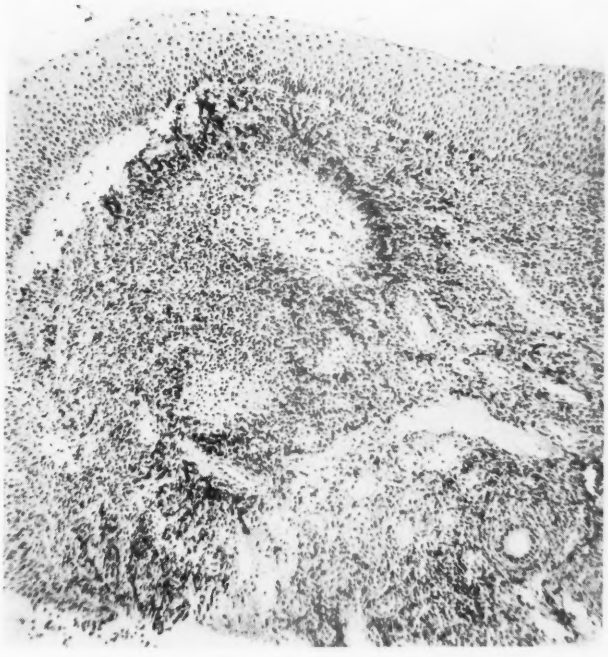

Fig. 4. Hyperplasia of pharyngeal lymph nodule in a child. (X72.)

base of the skull to the lower border of the larynx, near the level of the top of the seventh cervical vertebra. It is necessary, therefore, to inspect not only the oropharynx but the nasopharynx and laryngopharynx as well.

The normal mucous membrane of the pharynx is smooth excepting for the minute elevations which represent aggregations of lymphocytes. In the lower portion of the pharynx, the mucous membrane tends to be thrown into folds. The stratified ciliated columnar cells of the nasal fossae are continued into the nasopharynx and extend laterally to include the openings of the eustachian tubes. Most of the posterior wall of the pharynx, even the portion extending to the vault is covered by stratified squamous epithelium. Thus the oropharynx, including the lateral pharyngeal bands, and the laryngo-

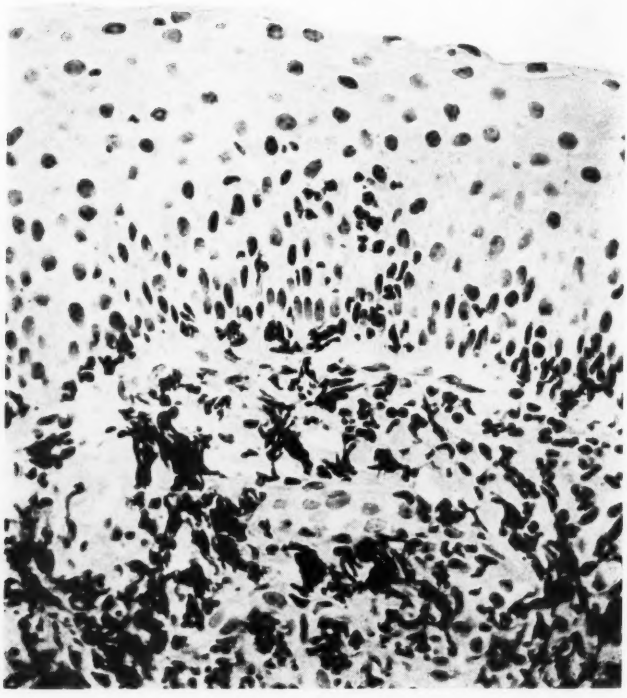

Fig. 5. Invasion by fibroblasts and early vacuolation of epithelial cells. Proliferation of endothelial cells in the peripheral lymph spaces. Pharyngeal tissue from a child with articular pains. (X306.)

pharynx is invested with this type of epithelium. Areas covered with ciliated epithelium appear redder, thicker and contain more glands but fewer lymphoid nodules and papillae than the areas covered with squamous epithelium.

The stratified squamous epithelium is composed of cells which stain sharply and distinctly. The basal layer of cells is clearly delimited by the basement membrane. Vacuolation of cells, distinct formation of papillae, blood vessels, and the presence of other cells including polymorphonuclear leucocytes are never found in normal epithelial tissue.

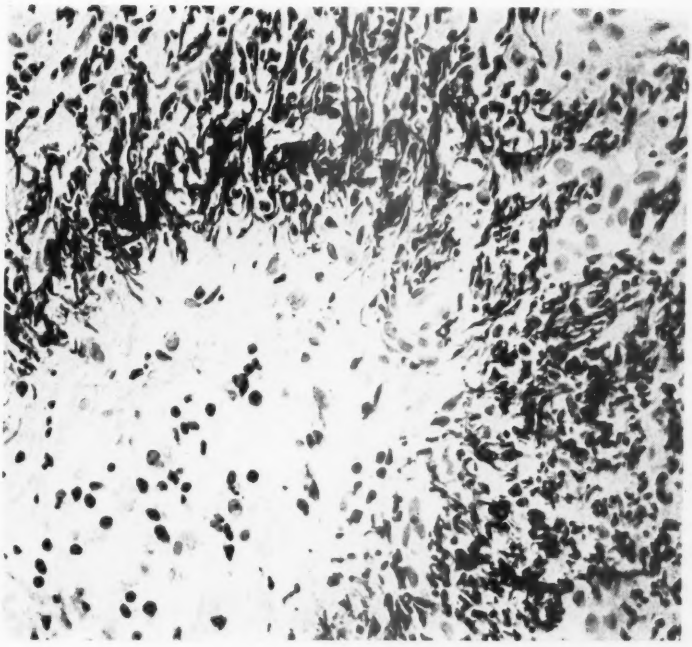

Fig. 6. Pharyngeal tissue from an adult (X306). The lymph cells have disappeared from the original site of the nodule excepting for a few cells which are irregular in size and in staining reaction. A dense zone of young fibrous tissue cells surrounds the nodule, obliterating the peripheral lymph channels and containing nuclear fragments.

Since the pharynx undergoes frequent alteration in position during movements concerned in phonation, respiration, deglutition and coughing, the mucous membrane is subjected to alterations in surface contour and in tension. The especially loose arrangement of the cells in the submucous layer provides a resilient structure to meet these requirements. The tunica propria is composed of fibro-elastic connective tissue supporting spindle-shaped or stellate connective tissue cells. Lymph spaces occur between the bundles of stroma tissue and are distributed in great profusion.

The number of solitary lymph nodules varies greatly in different individuals, but their structure is normally quite constant. They occur immediately beneath the basement membrane of the epithelium and are separated from it by intercommunicating lymph channels and

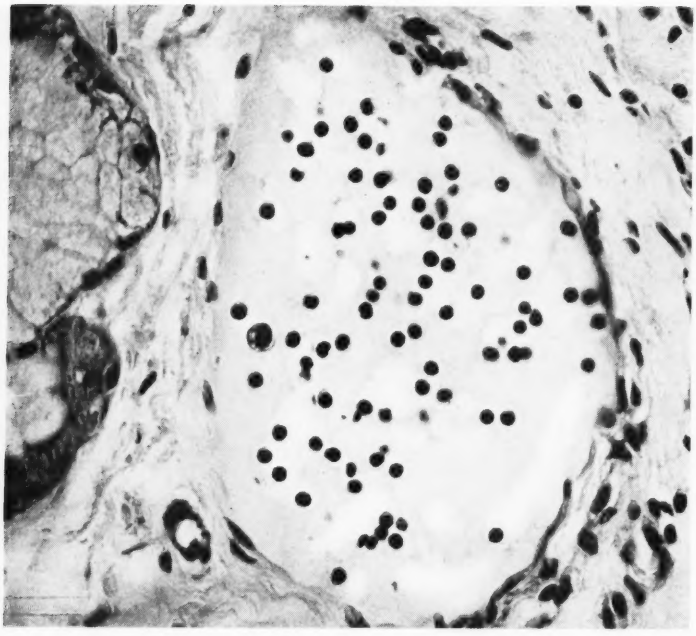

Fig. 7. Pharyngeal tissue from a child (X436). Dilatation of a lymph channel due to the combined obstruction by fibrosis about the channel and proliferation of endothelium within the channel.

a few tenuous strands of stroma tissue. The nodules are free from peripheral fibrous tissue so arranged as to constitute a capsule but are surrounded on all sides by lymph channels. Each nodule appears to consist almost exclusively of lymphocytes because, although not densely packed, these cells predominate to the extent of obscuring other cells. The lymphocytes are uniform in size and in staining reaction. Mitotic figures are rarely found. Polyblasts are sparse, and plasma cells or eosinophils rarely observed. The fibrous tissue stroma is inconspicuous.

Mucous glands are most numerous in the upper portions of the pharynx and in the region of the lateral pharyngeal bands. They occur at considerable depth, partly within the submucous tissue and partly between the muscle bundles. Their position is therefore well below the level of the lymph nodules. No constant relation between

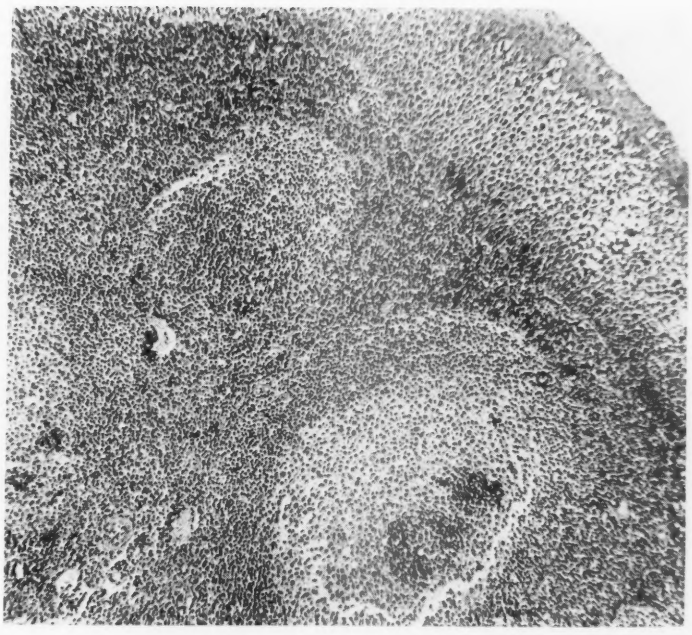

Fig. 8. Pharyngeal tissue from a young adult (X102). Marked hyperplasia of the lymph structures and chronic inflammatory changes. Degeneration and invasion of the epithelium.

the ducts of the glands and the lymph nodules can be demonstrated. The alveoli of the mucous glands are uniform in size and contain crescentic groups of cells resembling demilunes which appear to be aggregations of serous cells.

Histologically, certain specific tissue changes accompany chronic infection in the pharynx. These consist mainly of alterations in the lymphatic structures, and the solitary lymph nodules exhibit first marked hyperplasia and, later, changes which are characteristic of chronic inflammation.

The earliest change to be noted in the nodules is a numerical increase of lymphocytes. The progressive hyperplasia extends far beyond the original bounds of the nodule, approaching the muscle bundles below and altering the contour of the surface epithelium above.

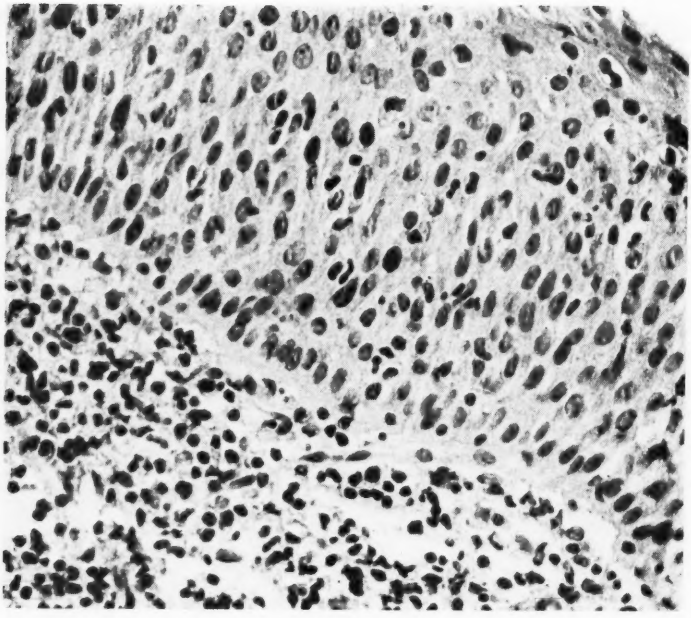

Fig. 9. Pharyngeal tissue from a child (X436). Early invasion of the epithelium by polymorphonuclear leucocytes. The peripheral lymph spaces are not as yet obliterated and there is little proliferation of fibrous tissue elements.

Once hyperplasia is established, fibrosis appears. Young fibrous tissue cells first appear on the border of the lymph nodule which is nearest the epithelium. As they increase in number they gradually obliterate the peripheral lymph channels and thus cause dilatation of the contiguous lymphatics. The obliteration of lymph channels is proportional to the degree of fibrosis but is accentuated by an accompanying proliferation of endothelium within the channels. The dilated lymph vessels contain numerous lymph cells and occasional plasma cells.

Further infiltration of young fibrous tissue cells gradually invests the original site of the nodule, and later, band-like extensions of fibrous tissue cells invade the surrounding accumulations of lymph cells to form pseudo-nodules. Eventually, the fibrous tissue bands contract to form a dense capsule.

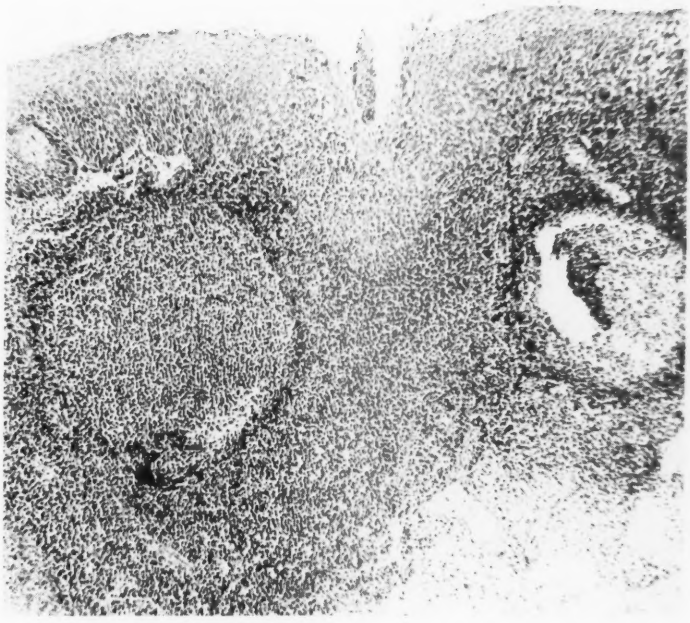

Fig. 10. Pharyngeal tissue from a young adult (X72). Evident hyperplasia and chronic inflammation with loss of the integrity of the basement membrane of the epithelium.

The cells of the lymph nodules have not achieved a high degree of differentiation, and they readily undergo mitosis and rapid proliferation when deleterious agents gather in the recesses of the nodule. Rapid proliferation not only occurs early in the course of the infection but is accompanied by an impressive increase in the number of polyblasts, indicating a stimulation of or attraction for the reticuloendothelial units. The increase of the number of polyblasts as well as the mitosis of the lymphocytes in the central portions of the nodule may give the appearance of a false germinal center.

Hyperplasia of the lymph nodules invariably appears to precede the proliferation of fibrous tissue. With the increase of fibrous tissue elements, however, a gradual and commensurate disappearance of lymphoid elements is noted. Nuclear debris in large amounts be-

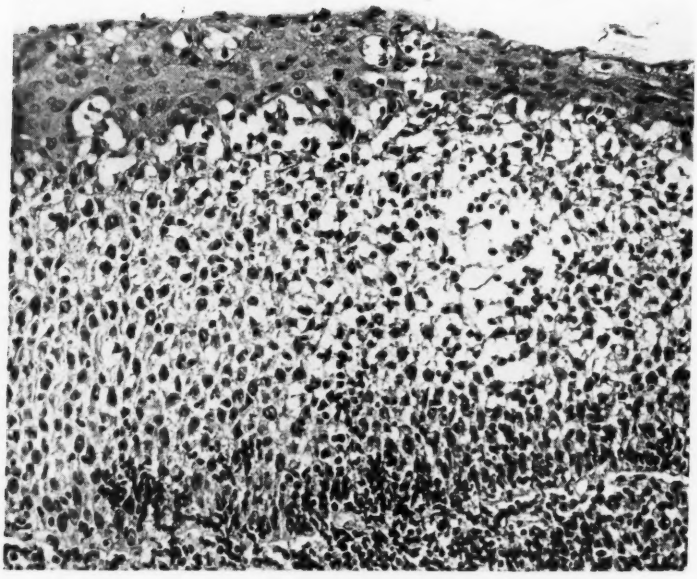

Fig. 11. Pharyngeal tissue from a child (X306). Vacuolation of the epithelial cells, with profuse infiltration of cells arising beneath the basement membrane.

comes evident, the greater concentration of the debris being contiguous to the fibrous tissue. Prior to the general dissolution of cells, the decrease in the number of lymph cells is coincident with and proportional to the increase in the number of polyblasts. Eventually, even the polyblasts disappear in the face of continued fibrosis, and the central portions of nodules and pseudo-nodules consist of material resembling hyaline degeneration.

Plasma cells do not appear to play an important role in these changes and eosinophils are only occasionally found. No giant cells appear. Polymorphonuclear cells appear quite early between the nodule and the basement membrane, and while they soon invade the epithelium in small numbers, they are sparse within the nodule and the submucosa.

With the earliest appearance of lymphoid hyperplasia, polymorphonuclear leucocytes penetrate the basement membrane and in-

vade the epithelium. Fibroblasts appear later, obliterating the space between the nodule and the basement membrane and finally invading the deeper layers of the epithelium. Extensive vacuolation of the epithelial cells follows with considerable attrition so that one or two layers of cells may be all that remain of a structure which is usually from 15 to 20 cells in thickness. In spite of this, ulceration is practically always absent. The infiltration of fibroblasts in chronic inflammation induces irregularities in the arrangement of the basement membrane which later become actual invagination of the surface epithelium. Such invaginations may occasionally possess a lumen filled with amorphous debris and desquamated cells.

Crypts are infrequently noted in the solitary lymph nodules of the posterior wall of the pharynx but are numerous in the region of the lateral pharyngeal bands. The appearance of numerous crypts in the follicles of the posterior wall accompanies hyperplasia of the lymph nodules and may be regarded as an abnormal finding. The contents of such crypts vary greatly. Thus the lumen of the crypt may be filled with leucocytes or it may contain merely amorphous debris. It is of some significance that the walls of the crypts may be imperfect or even denuded of epithelium.

In chronic inflammation of the pharynx the mucous glands become more conspicuous due to a numerical increase of alveoli without other obvious change. Frequently conspicuous lateral pharyngeal bands owe their increase in bulk to such hyperplasia of the glandular elements rather than to hyperplasia of the lymph tissue. The concomitant hyperplasia of the glandular structures is due to chronic inflammatory changes in the vicinity of the glands. No cellular invasion of the alveoli is found, and the lack of cellular infiltration in the alveoli and in the ducts of glands is in decided contrast to the condition of the crypts. Later, when fibrosis becomes a conspicuous feature of the reaction to chronic inflammation, the mucous glands undergo atrophy, and the secretions become sparse and frequently abnormal in consistency.

Endarteritis is commonly found, and newly formed endothelial cells occur in the meshes of the reticular tissue between the blood vessels. Margination of leucocytes in blood vessels adjacent to crypts is repeatedly observed. With the aging of the fibrous tissue, constriction occurs so that dilatation of some vessels ensues, but in many instances vascular atrophy and drastic reduction of vascular structure follow.

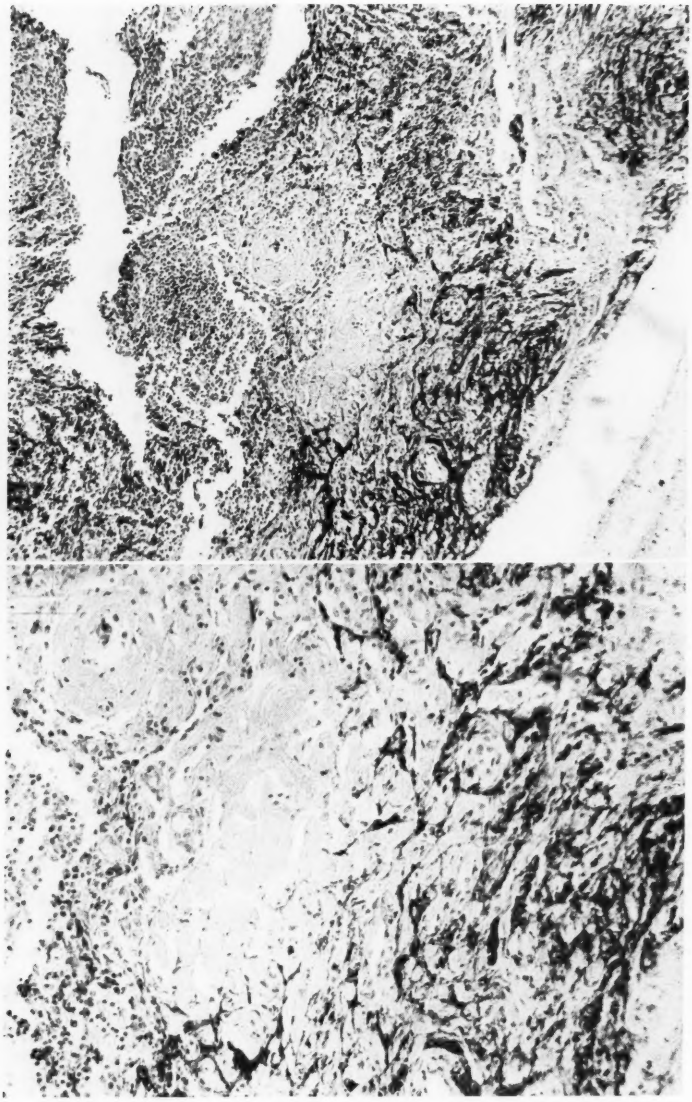

Fig. 12. Pharyngeal tissue from a child (A, X72; B, X280). Progression of fibrosis to the extent of obliterating other cellular elements. Hyaline masses represent the former position of the lymph nodules.

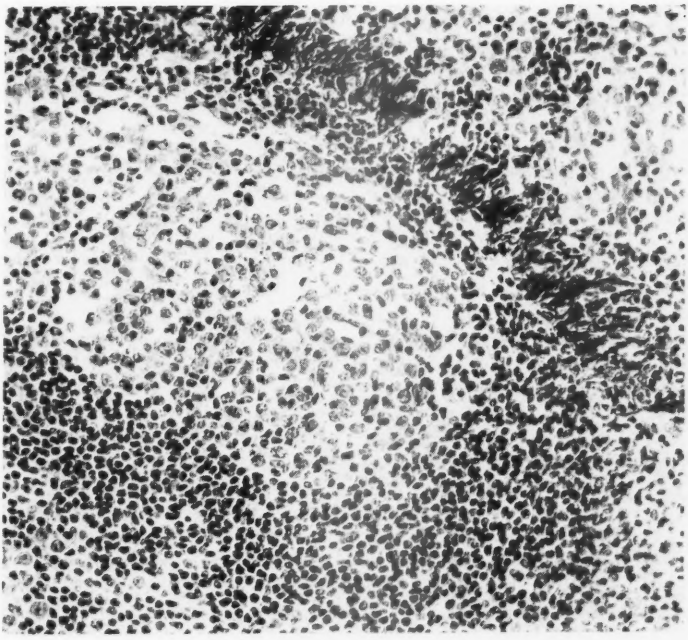

Fig. 13. Pharyngeal tissue from a child (X280). Definite invasion of the epithelium. Infiltration of young fibrous tissue cells between the nodule and the basement membrane of the epithelium, and extension of both fibroblasts and lymphocytes into the epithelium. Mitotic figures occur in cells distributed in the paler areas.

Histologically a specific tissue change is discovered that is characteristic of chronic infection in the pharynx. The extent of these alterations is so marked and so constant that the clinician cannot underestimate its significance. Regardless of the type of accompanying systemic manifestation, the lymph nodule is the principal seat of pathology, and marked hyperplasia with chronic inflammatory change is the rule.

An important role in the infection and reinfection of the lymph tissue of the pharynx is played by the sinuses. Posterior drainage from the ethmoidal and the sphenoidal sinuses bathes the pharyngeal wall and alters its structure. Contact with secretions from the lower

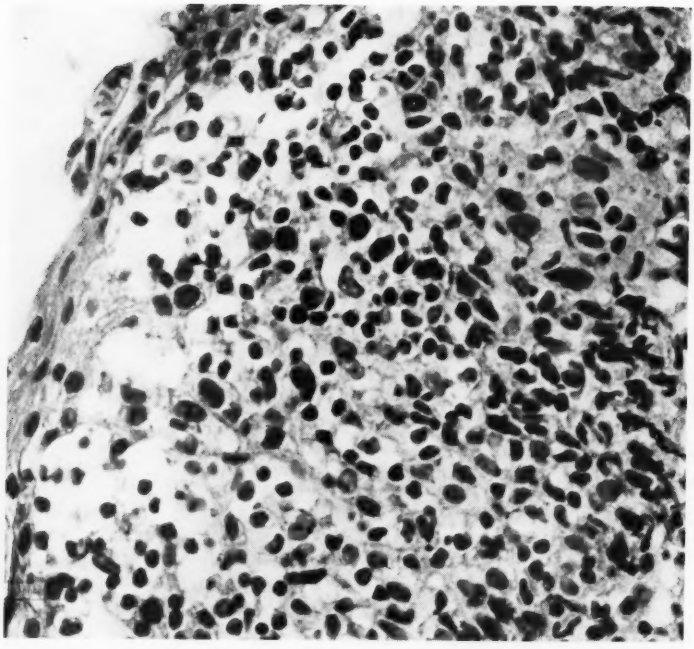

Fig. 14. Pharyngeal tissue from a young adult (X566). Attrition of the epithelium occurs without true ulceration.

respiratory tract, especially when continuous and forceful cough is a feature of the lower respiratory infection, is a not infrequent cause. Nasal obstructions are important predisposing factors in infection of this lymph tissue both before and after tonsillectomy. Inflammatory changes may follow otherwise transient acute infections in the oral cavity or nasal passages. But whether the infection arrives from above or below, the pharyngeal alterations continue long after the sinus or bronchial infections have disappeared.

In children, local subjective symptoms are usually absent. Adults are apt to attribute the local symptoms to catarrh or sinus disease, and the hypersecretion of the stimulated mucous glands may well be mistaken for drainage from the posterior sinuses. A dry hacking cough and even slight hoarseness may be noted during the

more active phases when the secretions are profuse, tenacious and thick. The pharynx may be generally injected and coarsely nodular or velvety, but as the glandular functions are impaired it becomes dry and glazed. Increased hyperemia accompanies the initial leucocytosis and continues until the fibrosis becomes sufficiently marked to influence the calibre and arrangement of the blood vessels. Lymphatic cell aggregations become augmented by additional cells until they appear elevated above the contiguous membrane. Retained debris within crypts is not always grossly visible. Enlarged blood vessels extend across the posterior wall of the pharynx and along the posterior pillars. While the injection of the mucous membrane is impressive because of its patchy distribution, the striking feature is the lymphoid hyperplasia, the nodules at times becoming almost polypoid. In prolonged cases the fibrosis and pallor of scarring are interspersed between injected areas. The mucous glands and lymph nodules behind the posterior pillars of the fauces are especially conspicuous because both glands and follicles are more closely grouped along the lateral walls. Nodules exhibiting extreme pallor usually show more marked histological changes than do the injected nodules. The absence of injection does not indicate quiescence of the infectious process or of the tissue changes.

The gross appearance of the pharynx in chronic infection varies greatly according to the duration of the infection. Any attempt to classify such chronic infection into types or groups results in an entirely artificial classification.

A clear understanding of both the gross and microscopic changes involved in infection of the extratonsillar lymphoid tissue of the pharynx is essential for the adequate treatment and eventual elimination of infection in Waldeyer's ring. The recognition and eradication of such infection assume major importance in those patients with obscure conditions attributable to focal infection and in whom no other demonstrable focus can be found. Failure to obtain complete control of infection in all portions of Waldeyer's ring has led to sufficient misconception and confusion as to discredit the concept of focal infection. It is, therefore, of extreme importance that the pathology and distribution of these lymphoid structures be generally recognized and appreciated in order that future clinical studies may be carried out on a sound basis.

1912 Spruce St.

LXI

CLINICAL CONDITIONS ARISING FROM ANOMALIES OR MALDEVELOPMENTS OF THE BRANCHIAL ARCHES AND CLEFTS*

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Various interesting clinical conditions result from anomalies or maldevelopment of the branchial arches and clefts. This report is a résumé of the material which has accumulated in the Lahey Clinic, along with a few cases which have come to my personal attention. These include: (1) variations in the right inferior laryngeal nerve (recurrent laryngeal nerve) and the great vessels in the upper chest and neck; (2) epidermal inclusion cysts or dermoid cysts; (3) preauricular sinuses, supernumerary cartilages about the ear, maldevelopment of the auricle, external auditory meatus and middle ear; (4) lateral cervical branchiogenic sinuses and cysts, and (5) thyroglossal tract anomalies and aberrant thyroids.

Congenital cleft lip and palate and congenital malformations of the external ear also fall into this group of cases. They will not be included in this discussion because, to be of value, these subjects must be considered in detail and they are now covered in work on plastic surgery. For anatomical and developmental details reference should be made to works on anatomy and embryology. Only slight mention will be made of the developmental process as required by the text.

ANOMALIES OF THE RIGHT INFERIOR LARYNGEAL NERVE, INNOMINATE ARTERY AND AORTA

Anomalies of the right inferior laryngeal nerve, the innominate artery and the aorta (Fig. 1) rarely present clinical problems requiring the services of the laryngologist. Figure 2 represents the conventional schematic development of the aortic arch and the great arteries of the upper chest and neck, and also of the larynx and the

*From the Department of Otolaryngology, The Lahey Clinic.

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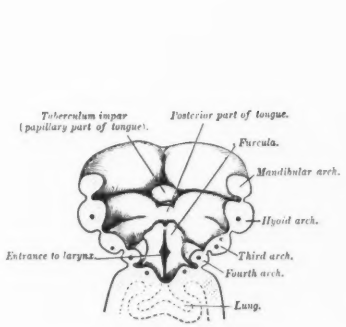

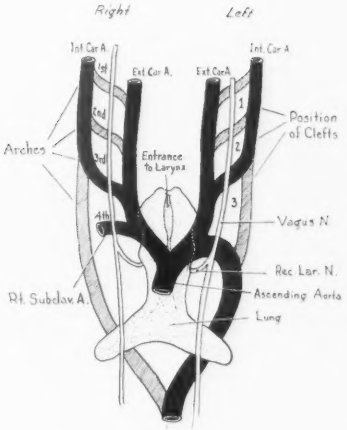

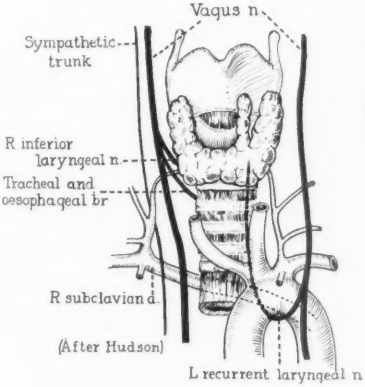

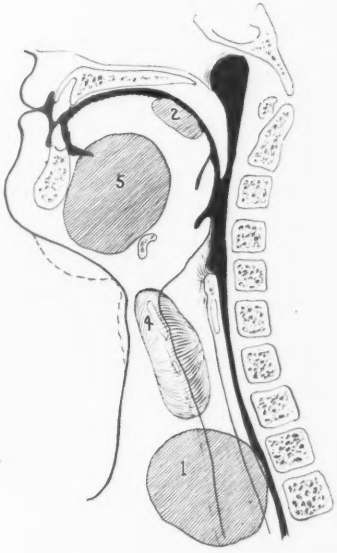

Fig. 1. The floor of the pharynx of a human embryo of about 23 days (after His), showing the close relation of the fourth branchial arch to the larynx.

Fig. 2. Conventional schematic drawing showing the development of the aortic arch and the great vessels of the upper chest and neck from the dorsal and ventral arteries, and the connecting arteries of the branchial arches. The dark vessels become permanent, the shaded vessels disappear. Superimposed is a drawing of the embryonic larynx with the vagus and inferior laryngeal nerves. In the adult the distance between the third and fourth arches becomes greatly elongated as compared to the early embryonic position.

Fig. 3. A diagrammatic drawing of a case of anomalous origin of the right subclavian artery, together with absence of a right recurrent laryngeal nerve. The right inferior laryngeal nerve proceeds directly from the vagus to the larynx (after Hudson). The right subclavian artery arises from the descending aorta on the left side and passes behind the trachea and the esophagus to the right side.

Fig. 4. An outline drawing representing the position of dermoid or epidermal inclusion cysts discussed in the text. 1—A cyst in the upper thoracic aperture; 2 the position of two dermoid cysts in the foramen of the tongue; 4 the relative position of four lateral cervical dermoid cysts; 5 the position in the median line of five epidermal inclusion cysts of the floor of the mouth.

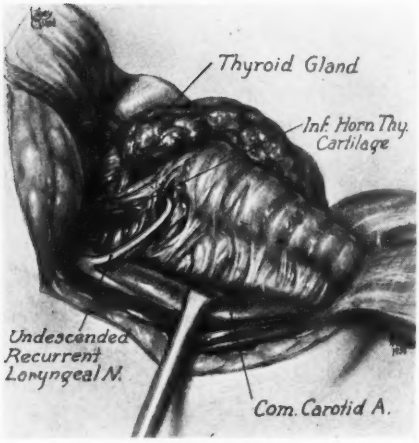

Fig. 5. A drawing made during a thyroid operation showing the direct passage of an inferior laryngeal nerve from the vagus to the larynx.

Fig. 6. *A*—The lateral view of a patient with an epidermal inclusion cyst of the floor of the mouth which protrudes downward, producing a prominence below the chin and pushing the tongue upward. *B*—A case with a somewhat similar picture, with a cystic hygroma, presented as of value in differential diagnosis.

laryngeal nerve, showing the close relation of the larynx to the fourth branchial arch where the inferior laryngeal nerve (later the recurrent laryngeal nerve) innervates the larynx by crossing below the artery of the fourth branchial arch. The later separation of the larynx and the great vessels produces the recurrent portion of the inferior laryngeal nerve.

With the anomalous origin of the subclavian artery as described by Hudson and others (Fig. 3), there is no recurrent nerve on the right side but a direct connection between the vagus and the larynx by the inferior laryngeal nerve. Two such cases have been observed at the clinic during thyroid operations (Fig. 5).

The knowledge of this variation may prevent injury to this nerve during dissections in the neck and during thyroid operations. This variation may account for some otherwise unexplained paralysis of the inferior laryngeal nerve on the right side. G. D. Williams examined 159 aortic aches and found four anomalous right subclavian variations. He suggested that a surgeon may find one or more of these cases in every 100 thyroid operations that he performs.

The anomalous right subclavian artery crosses behind the esophagus and in rare instances presses upon the esophageal wall with the production of a deformity of the barium column as seen in the fluoroscope, and in one case was verified by esophageal examination. This patient complained of a sensation of solid foods sticking in the throat.

In rare instances the distal portion of the artery of the right fourth arch may persist and a bifid aorta result, with the trachea passing through or between the two portions of the aorta. Such a sensational case was presented from the Massachusetts Eye and Ear Infirmary before its staff meeting, and before the New England Otological and Laryngological Society, by the service of Dr. Schall. The child had difficulty in breathing during a respiratory infection. A tracheotomy was done, without relief, but the passage of a bronchoscope low in the trachea gave a good airway. After a number of bronchoscopies in an attempt to keep the trachea open, a long tracheal tube was placed, which afforded an excellent airway. In the course of several days the patient died of a profuse hemorrhage. The postmortem examination revealed a bifid aorta through which the trachea passed and by which the trachea was greatly narrowed. Erosion of the aorta produced the hemorrhage.

A rigid tracheal tube is prone to erode the wall of a vessel after a period of several days or weeks when it is placed in the trachea to relieve tracheal compression from a great vessel of the thorax or the

root of the neck. I have seen two cases of lethal hemorrhage follow erosion of the vessel walls under these circumstances.

EPIDERMAL INCLUSION OR DERMOID CYST

Epidermal inclusion or dermoid cysts may occur at the line of fusion, that is, along the median line of the tongue or jaws, or may be found in the anterolateral aspect of the neck from the arch of the aorta to the angle of the jaw (Fig. 4). I have seen five median line inclusion cysts in the floor of the mouth. These cysts were large, varying in size from an egg to an orange, smooth and located in the median line (Fig. 6A). The cysts were attached to the median raphe and were well encapsulated and easily separated except at the attachment to the median raphe. A cystic hygroma shows a similar external picture (Fig. 6B).

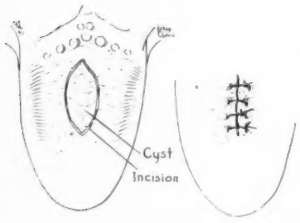

Fig. 7. An outline drawing showing the position of the dermoid cyst on the dorsum of the tongue, the region excised and the method of suture.

The only tumor which I have confused with these cysts was a large neurofibroma which was located in exactly the same position and gave a characteristic picture. I did not realize I was dealing with a solid tumor until removal was begun which revealed no attachment to the median raphe and a lobulated under surface.

These cysts have been treated by complete removal through a transverse sublingual incision just above the submaxillary ducts. Due to the large size of the cysts, it may be necessary to evacuate the cyst partly, to deliver it through the mouth and separate it from its posterior attachments along the median line. Following removal, the bleeding is controlled and the cavity largely obliterated by a few sutures. A drain is placed in the cavity, and the lateral margins of the incision only are sutured. In all five cases so treated the patients made rapid recovery and recurrence has not taken place. These cysts did not contain hair but had typical sebaceous material in them.

Two true dermoid cysts have occurred in the median line on the dorsum of the tongue anterior to the foramen cecum (Fig. 7). One patient brought in a hair which he had pulled from the back of his tongue. He also had recurrent swelling and tenderness of the tongue. In the second patient there was likewise recurrent infection

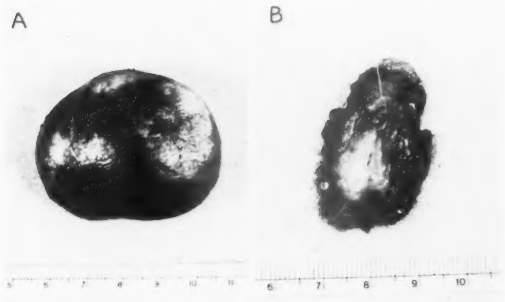

Fig. 8. *A*—A photograph of a dermoid cyst removed from beneath the tongue. The cyst was well encapsulated and quite smooth. *B*—A dermoid cyst from the dorsum of the tongue. The cyst has been slit open, showing the epithelial lining and a coiled-up hair in the cyst.

with the discharge of pus in the region of the lower pole of the right tonsil. Following subsidence of the acute inflammatory process, an indurated area was definitely located on the dorsum of the tongue in the median line. The treatment in each case was complete removal, using a transnasal intratracheal tube for anesthesia, packing the pharynx and, with sharp dissection, removing an oval-shaped area of tongue large enough to include the entire indurated area containing the dermoid cyst. The wound was closed and bleeding controlled by sutures (Fig. 8).

Four dermoid cysts have been found on the anterolateral aspect of the neck. A definite preoperative diagnosis was not made in these cases, the true diagnosis having been made only after the cysts were opened. These cysts contained hair as well as sebaceous material, and the history points to their presence for a long time. In most cases a branchiogenic cyst was suspected.

Differential diagnosis includes any mass in the neck, and these will be enumerated under the branchiogenic cysts and sinuses. The treatment is complete surgical removal, and the technic is the same as that used for benign tumors in this region.

In one case of special interest the dermoid cyst was located in the upper thoracic aperture (Fig. 9), and had been asymptomatic until the patient, a girl of 13, began her first menstrual period. At this time a deep, firm tumor appeared in the upper thoracic aperture

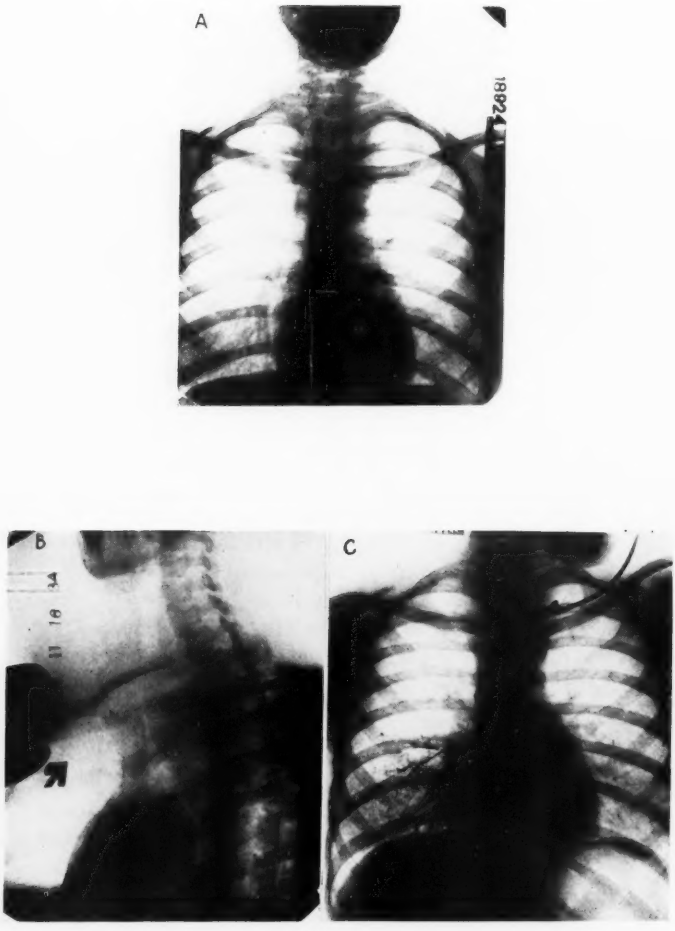

Fig. 9. *A*—Roentgenogram anteroposterior, showing tumor of the upper thoracic aperture with the trachea pushed to the right and greatly narrowed. This tumor proved to be a dermoid cyst of the upper thoracic aperture. *B*—Lateral view of the same patient showing the tumor posteriorly pressing upon the trachea, pushing it forward. *C*—The same patient after the dermoid cyst had been opened, with the injection of lipiodol into the trachea and into the cyst to show the position of each.

obstructing the trachea; an emergency tracheotomy was necessary. A cystic tumor with hemorrhage into it was suspected, and an incision was carried down to the tumor and the tumor opened. It proved to be a thick-walled dermoid cyst. The opening of the cyst relieved pressure, and at the second procedure the entire dermoid cyst was dissected out. The patient made an uneventful recovery.

PRE-AURICULAR SINUSES

Pre-auricular sinuses are frequently found as only a small depression anterior to the helix (Fig. 10). They are asymptomatic or the symptoms are extremely mild and do not require medical or surgical attention. A shallow sinus may fill with desquamated epithelium which requires expression of debris similar to a blackhead. Sinuses complicated by recurrent inflammation increase in size, become deep, contain granulations and may produce profuse discharge. The irritant discharge may further produce an unsightly area of dermatitis about the ear.

The history of those requiring surgical attention usually reveals recurrent acute infection with periods of quiescence with only mild symptoms intervening. During the acute infection some have been incised and curetted. In the interval others have been operated upon, with some having had repeated unsuccessful attempts at removal. Some advanced cases may go for a considerable period of time with active infection and little tendency of the symptoms to abate.

The location of the fistulas which have been treated surgically is as follows: four immediately in front of the ear at the crux of the helix, two below and anterior to the tragus over the jaw, and two which began, no doubt, in the region of the crux and extended upward into the hair and backwards, and another which extended not only in these directions but behind the ear and about the external auditory canal as well.

In the first case that I treated surgically I injected the area with methylene blue to stain the tract. In my dissection I failed to get the entire tract and the patient continued to have symptoms, but she would not submit to further surgical excision. However she would come for observation or would call me with each acute exacerbation. Since that time the treatment practiced by us has been the appropriate use of antiseptics and hot fomentations and supportive treatment during the acute inflammatory stage, followed by the complete surgical excision of the fistula along with all the involved subcutaneous area. The injection of dyes has been discarded. Bleeding

is controlled and the skin is sutured, with small rubber dam drains. In my experience, the infected tract has not extended through the temporal fascia. The sinuses anterior and inferior to the ear have been deeper, and deep in these fistulas aberrant cartilage has been found. In the cases of sinuses anterior and inferior to the ear the possibility of injury to a branch of the facial nerve must be considered. In our two cases no paralysis of any muscles resulted. Of the nine cases treated surgically one was a failure and in eight, complete cures have resulted.

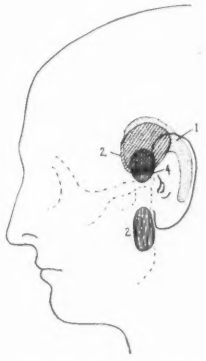

Fig. 10. Outline drawing showing the position and the number of the pre-auricular sinuses occurring in patients who have been treated surgically in the clinic.

SUPERNUMERARY CARTILAGE

Supernumerary cartilages may be unsightly (Fig. 11A). They are easily removed by excision of the cartilage well below the skin surface and suturing the skin in place. Five such patients have submitted to operation, and the results have been excellent.

MALDEVELOPMENT OF THE AURICLE, EXTERNAL AUDITORY CANAL AND MIDDLE EAR

Maldevelopment of the auricle, the external auditory canal and middle ear often are associated (Fig. 11B). The details of plastic repair of the external ear are in the field of the plastic surgeon. When the subject is a male and is backward or sensitive about his deformity, certainly he is entitled to the services of a plastic surgeon. Women may well hide their deformities with their hair. To date my experiences have been only with unilateral deformities. In these unilateral cases the hearing of the normal ear has been good, and therefore there has been no need for surgical interference so far as an aid to hearing is concerned. In cases of bilateral deformities with a functioning labyrinth, such a procedure may be justified. In one case, mastoid infection occurred and a radical mastoid operation was done, followed by the skin graft to the cavity. In this case the new external auditory meatus now remains wide open, but the hearing has not improved.

BRANCHIOGENIC SINUSES AND CYSTS

Lateral cervical branchiogenic sinuses or fistulas may be classified into three types: (1) the complete fistula which has both an

external opening above the clavicle, lateral to the median line, extending upward and laterally, and an internal opening into the pharynx near the lower portion of the tonsillar fossa (Fig. 12); (2) the incomplete external fistula which has only the external opening, and (3) the incomplete internal fistula which has an opening only into the pharynx.

Branchiogenic cysts may be found anywhere along the tract transversed by the complete branchiogenic fistula. A symptomless small depression in the skin above the clavicle may be the only finding, or small tracts may produce a small amount of glary mucoid discharge. Several of these cases with minor symptoms or no symptoms have been seen but not subjected to surgical intervention.

Excision has been done in 27 patients who have had severe enough symptoms to warrant surgical intervention (Fig. 13). In these cases the symptoms have been those of a fistula, with intermittent discharge or continuous discharge with dermatitis, acute recurrent inflammatory attacks with purulent discharge either into the mouth or onto the skin of the neck and a tumor in the neck. The most common complaints are discharging fistula, recurrent acute inflammation and tumor. While the condition is of congenital origin, symptoms that require surgical intervention may not develop until late in life. Of these patients on whom operation was performed, 56% were over 30 years of age, and 70% were females.

The differential diagnosis includes all tumors or masses that can be found in the neck. The following are the most common lesions from which branchiogenic cysts and sinuses must be differentiated: cervical adenitis from any infection, tuberculosis, syphilis, cervical abscess, actinomycosis, Hodgkin's disease, lymphosarcoma, lymphatic leucemia, dermoid cysts, thyroglossal cysts and sinuses, cystic hygromas, cavernous hemangiomas, lipomas, fibromas, neurofibromas, myomas, carotid body tumors, metastases to the glands of the neck from malignant tumors elsewhere, and aberrant thyroid. A definite preoperative diagnosis was made in only 57% of the cases which were treated surgically.

The treatment followed in the clinic has been complete surgical excision. In lesions that are uncomplicated by infection or previous surgical intervention, the cyst or fistulous tract usually is readily separated from the surrounding tissue. Thirty-four per cent of the patients operated upon had had previous surgical or roentgen treatment before they came to the clinic. When acute infections are present, operation should be delayed until the acute symptoms have

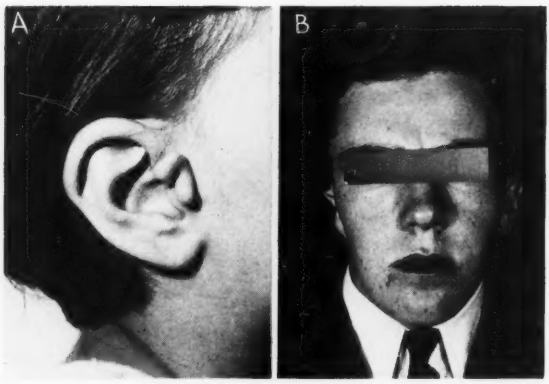

Fig. 11. *A*—A girl of seven with a supernumerary cartilage anterior to the ear. *B*—A fairly typical maldevelopment of one auricle. The hearing is good in the normal ear.

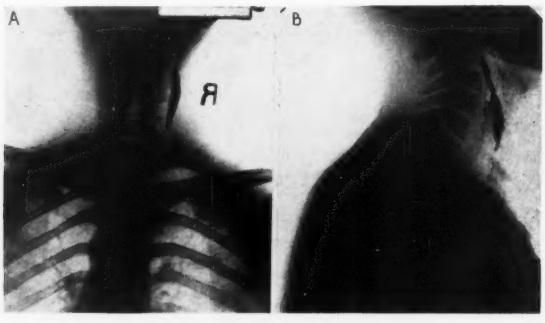

Fig. 12. *A*—A roentgenogram of a complete branchiogenic fistula injected with lipiodol, showing the dilated fistulous tract beginning in the neck and ending in the pharynx. *B*—Same case as shown in *A*, lateral roentgenogram.

Fig. 13. *A*—Photograph of a man with a very large branchiogenic cyst developing fairly low in the neck. *B*—A large branchiogenic cyst in a woman, developing fairly high in the neck.

subsided. Both infection and previous surgical intervention may make the dissection more difficult.

In cases of complete sinuses the so-called step-ladder method of surgical excision has been employed. A transverse incision is made over the external opening (Fig. 14A) and a piece of skin with the sinus is freed up. Traction is made upon the fistulous tract, and the dissection is carried upward as far as this exposure permits. A second incision is then made under the jaw roughly parallel to the first, and the tract is brought upward and out the second incision. Care is taken in this dissection not to injure the great vessels. The upper incision allows adequate exposure up to the pharyngeal wall. The hypoglossal nerve is usually exposed in the upper dissection and must be preserved, and the sinus is sectioned at the pharyngeal wall without inversion of the stump into the pharynx. Such an incision leaves little scarring and gives adequate exposure. The incisions are closed with small drains. Only one incision is usually required with incomplete sinuses and cysts. The results in this series have been most satisfactory, without any recurrences.

THYROGLOSSAL TRACT AND ABERRANT THYROID

Normally the thyroglossal tract is obliterated completely before birth, the thyroid having descended from the foramen cecum in the tongue to its normal position in the neck. If the thyroid fails to descend, a lingual thyroid develops on the base of the tongue. Six cases of lingual thyroid have been observed in the clinic, three of which have been verified microscopically, one in a woman of 68, who died of leucemia, and in two, surgical excision has been accomplished. All six cases have occurred in women. Three of the cases were found on routine nose and throat examination, and were asymptomatic. The symptoms from the lingual thyroid depend largely on the size of the tumor, and in the cases above mentioned, have produced mainly difficulty in swallowing solid foods and interference with speech. In two cases the tumor ascended into the nasopharynx behind the soft palate on swallowing.

The diagnosis is usually very easily made because of the characteristic appearance and position of the tumor. It is firm, smooth and resilient to palpation. I have seen only a neurofibroma and a thyroglossal cyst which were confusing from the standpoint of differential diagnosis.

For treatment of lingual thyroid when symptoms are sufficient to make excision worth while, surgical excision is performed under

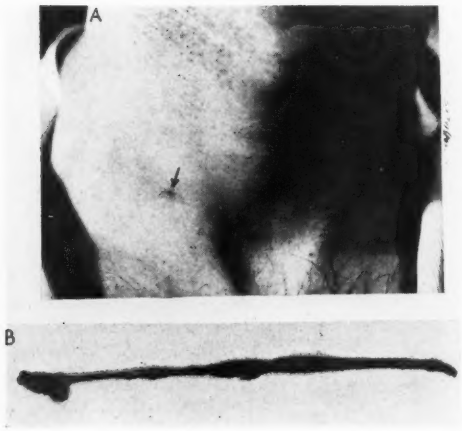

Fig. 14. *A*—The external opening of a complete branchiogenic fistula. *B*—A Photograph of the specimen removed from the same patient.

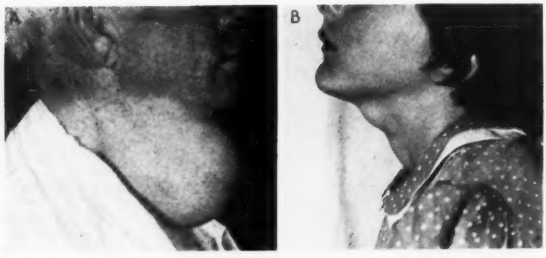

Fig. 15. *A*—A photograph of a very large thyroglossal cyst. A definite diagnosis was made only after excision of the cyst. *B*—A fairly typical photograph of a small cyst which had previously been opened; the patient was a young girl.

transnasal intratracheal anesthesia. The mouth is widely opened and strong traction is placed on the tongue, bringing the base of the tongue well forward into the mouth where it is fairly readily accessible. The main body of the tumor is removed by clamping and cutting. It is not necessary to remove the entire gland. Bleeding is controlled by sutures. Recovery is without incident. In this operative procedure there is no danger of removing the parathyroid glands. In the two cases of lingual thyroidectomy both patients required thyroid feeding. Thyroid deficiency to some degree is frequent in cases of lingual thyroid, due to the limited size and activity of the gland.

Following descent of the thyroid gland into the neck, if obliteration of the tract is incomplete, a cyst, sinus or fistula giving clinical symptoms may arise from the epithelium of the tract. Malignant change in the epithelium of this tract is rare. We have observed one case in which a highly malignant tumor occurred in the thyroglossal tract of a girl 19 years of age, which proved fatal even though the tumor had been partially excised and X-ray therapy had been given before the patient came under our observation and in spite of large doses of X-ray under our supervision. Adenocarcinoma was found in a second case and was entirely removed with the cyst. Epidermoid carcinoma arising in the thyroid gland most probably originates from thyroglossal tracts, and such a case has been reported from the clinic.

Cysts are by far the most common anomaly of the thyroglossal tract. They are a type of retention cyst, containing mucus and epithelial debris. These cysts are prone to acute inflammatory processes. While uncomplicated they may vary in size from a small asymptomatic midline tumor which is just perceptible, to the size of a grapefruit (Fig. 15). These tumors are fixed to the hyoid and rise on swallowing.

When complicated by infection these cysts or the area about them become red and tender. They may change rapidly in size, being distended with the inflammatory exudate, and may either rupture spontaneously or require surgical drainage. When the inflammatory process has subsided, the cyst may refill or a fistula may have been established with the discharge of glary mucus, either clear or milky in color, onto the neck. Rarely, a fistulous opening through the foramen cecum may develop through which the contents of the cyst may be discharged, or the cyst may be in the substance of the tongue itself.

Early in the experience of the clinic excision of the cyst alone or of the cyst and the body of the hyoid bone was performed, but

recurrences were frequent. However, since the technic reported by Sistrunk in 1920 has been followed, recurrences have been extremely rare. This method consists of the complete dissection of the cyst, following the tract to the hyoid bone, removing the central portion of the hyoid bone and excising a generous core of muscular tissue from the hyoid to the foramen cecum. To date 211 cases have been treated surgically.

LATERAL ABERRANT THYROID TISSUE

Lateral aberrant thyroid tissue has been shown by Norris to develop from the posterior portions of the third and fourth branchial pouches. Norris has demonstrated the fusion of these lateral bodies with the main body of the thyroid gland in a 15 mm. human embryo. Later these lateral bodies atrophy and take no part in the formation of the adult thyroid gland. The failure to atrophy accounts for the thyroid tissue in the lateral position. These may develop either unilaterally or bilaterally. In the adult they may be found any place along the sternomastoid from the clavicle to the mastoid process in front of or beneath the anterior border of this muscle. On palpation they are recognized as soft, movable, discrete gland-like structures, and are easily mistaken for lymph nodes. It is important to keep the possibility of lateral aberrant thyroid tissue in mind as they are papilliferous in character and prone to malignant change. Before malignant change has occurred, radical dissections of the neck from the clavicle to the mastoid should be done to guard against it. This should be followed by intensive X-ray radiation, since these masses are very sensitive to X-ray therapy. If malignancy has occurred, the same treatment should be prescribed. In the absence of malignancy the prognosis is excellent, and even when malignant change had occurred only a small percentage (two in our series) of the patients died of recurrence. Careful follow-up studies are necessary to observe possible recurrence. This possibility must be kept in mind in cases of apparent cervical adenitis, and biopsy is required to make the diagnosis. Thirty-six patients have been subjected to surgical and X-ray treatment to date.

SUMMARY

A number of developmental conditions of clinical importance to the otolaryngologist has been briefly discussed. The direct innervation of the larynx from the vagus by the inferior laryngeal nerve on the right side and the anomalous development of the right subclavian artery from the arch of the aorta on the left, crossing

behind the esophagus, with pressure upon it, occur frequently enough that laryngologists should keep the possibility in mind. Tracheal obstruction by an anomaly of the aorta is rare. Supernumerary cartilages about the ear and malformation of the ear are mentioned.

Our experience with a large number of dermoid cysts, thyroglossal cysts and sinuses, lateral cervical branchiogenic cysts and sinuses and pre-auricular sinuses is summarized. It should be emphasized that successful treatment of these cases requires complete surgical removal.

The problem of the lingual thyroid is well understood although it occurs rarely. A case of malignant tumor arising from the thyroglossal tract is reported. The tendency to malignant change in and the frequency of lateral aberrant thyroid are reviewed, and the importance of early diagnosis by biopsy is stressed.

LAHEY CLINIC.

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LXII

ENVIRONMENTAL SURGERY IN OTOLARYNGOLOGY

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MIAMI BEACH

Looking back over his contributions to the specialty, the writer notes that a considerable number have dealt with borderline subjects, some of which his confreres have thought were outside the field of otolaryngology. But he has never ceased to contend that the structures environmental to the ear, nose and throat should be included in the legitimate field of this specialty and that the best results from surgical treatment can come only when this course is persistently and intelligently followed. And having taken this position throughout his professional life he now fancies it may not be amiss to discuss the question briefly again in the Jubilee year of the ANNALS.

A much limited field was that of the laryngologist's own choosing at the founding of the specialty. No one had ever seen the interior of a living larynx or of any other deeply-seated cavity of the neck. It seems to have been a burning, unquenchable desire of these new specialists to acquire an art whereby they could not only see the inside of these spaces but, by developing a degree of skill heretofore unknown, could determine the nature of any disease which may have developed there and could apply medicaments directly; or in the event a growth were discovered they could remove it. There was no thought of major surgery, or indeed of surgery of any kind, except that which could be performed through normal pathways.

These early specialists were mostly learned men who, attracted by the apparent possibilities of the new specialty, had switched to it from another branch of medicine. They had had no training in laryngology except that which was self-directed. But many of the more enthusiastic and ingenious among them practiced the methods so assiduously that they developed an art of examination and treatment of the larynx that has always seemed to the writer almost unbelievable, so dexterous were their movements, so gentle their application. This art as exemplified by the best of those who at-

tained it will probably never again be equaled and certainly will never again be surpassed.

Otolaryngology soon became a popular and honored branch of medicine. It was believed by many that these enthusiasts would be able to relieve or cure most affections of this difficult region by an approach through entirely natural channels.

But the next generation of laryngologists was on the whole not of equally high standard. Many of them had but scant qualifications and almost no preliminary education, and had graduated from medical schools of the very lowest grade. Some of them had attended special courses in one of our great centers where the most popular mode of instruction was teaching over the shoulder. A few attended foreign clinics where the opportunities were but little better than at home and the results were often equally poor. Pathology, anatomy and the principles of surgery received but slight attention. The student reveled in what he called practical instruction, which when translated meant standing or watching from a distance the favorite methods of treatment advocated by the instructor. Removal of nasal polypi and crude operations on the tonsils and adenoids were frequently demonstrated, as were the methods of treatment with sprays.

Postgraduate students returned with a score or more formulae for making spray solutions. Almost equally common was the array of seductive, shining apparatus for producing the spray. It was also a period of fine offices, and the attractive apparatus brought many patients to receive the soothing sprays which sometimes contained habit-forming drugs. The specialists seemed too often satisfied with this mode of practice, and many continued to follow it throughout a long professional career. Gone was the marvelous dexterity that had been developed in previous years. Otolaryngology made no progress and fell into disrepute. Tonsillotomy and cauterization of the turbinates were extensively performed, but commendable surgery of any kind was rare. The specialty had reached its lowest level. Environmental surgery made no progress.

Fortunately the next generation was more progressive. A few otolaryngologists had never ceased to advocate and to practice the surgery of the environment and to urge thorough preparation for its performance. Mastoid surgery was well done in most cities, and surgery of the tonsils and accessory nasal sinuses was improved. But too few, as yet, had studied the basic subjects necessary to good environmental surgery.

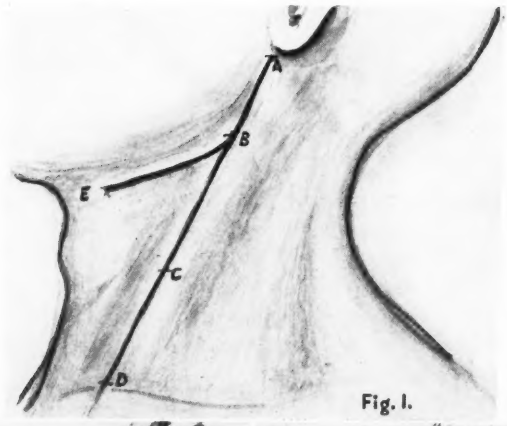

Fig. 1.

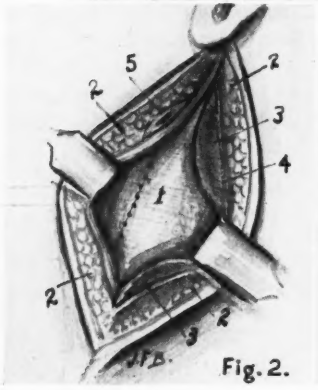

Fig. 2.

Fig. 3.

Fig. 1. Showing lines of incision for various operations on the neck. AB—For surgery of external and internal carotid arteries and lymph nodes of upper portion of deep cervical chain. AC—For ligation of internal jugular vein above common facial vein. AD—For block dissection of neck, ligation of common artery, removal of deep cervical lymph nodes. ABE—For operation on submaxillary lymph nodes and submaxillary salivary gland. The incision should be made along the anterior margin of the sternomastoid muscle and not over the muscle. In the event the tumor or abscess can thus be better operated upon, the line of incision may be along the posterior margin of the muscle. When made along the latter course, the position of the eleventh nerve must be recalled, for the nerve must be avoided. It is found at the junction of the upper and middle thirds of the sternomastoid muscle.

Fig. 2. Showing primary incision along the anterior margin of the sternomastoid muscle, down to the external layer of the cervical fascia. This fascia is continuous around the entire neck. Only the part which covers the upper portion of the muscle is shown. 1—Superficial layer of cervical fascia overlying sternomastoid muscle. The dotted line shows the anterior margin of the muscle where the incision should be made. 2—Skin and fat; 3—under surface of superficial fascia; 4—cut edge of superficial fascia; 5—external jugular vein cut across.

Fig. 3. Incision through soft structures of the neck along the upper anterior margin of the sternomastoid muscle. The arteries and internal jugular vein are shown as usually depicted, without any covering of fascia, giving the impression that these vessels run through the neck without any such covering. Figure 6 shows the situation with its coverings of skin, muscle and fascia as it really is after an incision of the carotid sheath has been made. 1—Skin and fat; 2—sternomastoid muscle from which superficial layer of deep cervical fascia has been removed; 3—superior thyroid artery; 4—external carotid artery; 5—internal jugular vein, the upper portion of which has been removed to show the artery; 6—bifurcation of common carotid artery; 7—omohyoid muscle; 8—hypoglossal nerve.

Meanwhile the more forward-looking members of the specialty saw the need for doing better and more extensive environmental surgery than had yet been attempted. These recognized the necessity for basic study and especially the need of acquiring a better knowledge of the anatomy of the special field. They eagerly sought opportunities for sound postgraduate study, and a few leading universities, having recognized the necessity for such study, established courses that met the wants to some extent, especially along anatomical lines.

There were also other helps for those looking to the betterment of the specialty. The American Board of Otolaryngology encouraged those who were keenly anxious to improve their status. American societies with high standards and a large combined membership were stimulating examples to those who wished to rescue laryngology from the low plane to which it had fallen. But the general surgeon continued to do much of the environmental surgery.

The "spraying epoch" was passing, and signs of a saner, broader field of surgery were visible. A few otolaryngologists were doing commendable operating. They traced the spread of infection, when occurring, from the middle ear or the mastoid antrum and performed an intracranial operation in the event a brain abscess had formed. They also recognized infection of a sinus of the dura when it followed an ear infection, and did not hesitate to do whatever surgery was necessary to remove an infected clot. The more daring ones even followed the infection into the internal jugular vein, in the event the thrombus had extended downward into the neck. Then, even though the affection had become environmental, they exposed the carotid sheath, slit open the vein, removed the thrombus and when necessary excised the affected portion of the vessel. These same otologists did not hesitate to enter the neck for the purpose of draining a deep abscess, Ludwig's angina, for example.

As previously stated, the early otolaryngologists were not surgically inclined. They limited their activities chiefly to skillful applications to the laryngeal interior and to such operative procedures as could be performed inside the nose or larynx. The general surgeon continued to do the environmental surgery. Having always been accustomed to operating on this area, he looked upon the new specialist as an unprepared trespasser.

But the accusation that all otolaryngologists were unprepared had lost much of its former weight. Those who had entered the specialty in recent years had had excellent ground-work in the schools, and their work in a hospital following graduation had pro-

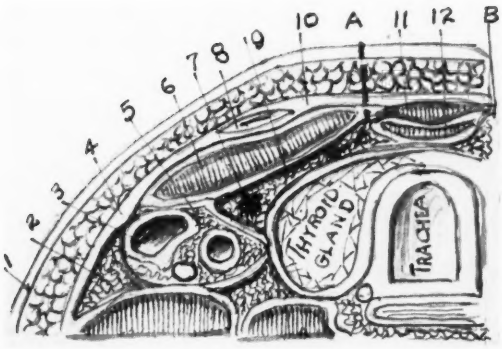

Fig. 4. Cross-section of neck showing how the fascia wraps up each separate compartment to protect the contents from affections that have origin in a connective tissue compartment. Pathway of safe approach to abscess is shown. 1—Skin and fat; 2—connective tissue compartment; 3—interior of carotid sheath, showing internal jugular vein, carotid artery and vagus nerve. Note that the sheath is surrounded by areolar tissue. 4 and 10—superficial layer of cervical fascia; 5—connective tissue inside carotid sheath; 6—sternomastoid muscle. Note that the two layers of fascia completely surround it, making a closed compartment. 7—Abscess in a connective tissue compartment; 8—external jugular vein; 9—connective tissue forming line of cleavage; 11 and 12—ribbon muscles enclosed by fascia. A—Line of incision down to anterior margin of muscle. There is but one layer of fascia here. Note that the incision through this fascia is in front of the muscle, the compartment of which should not be opened.

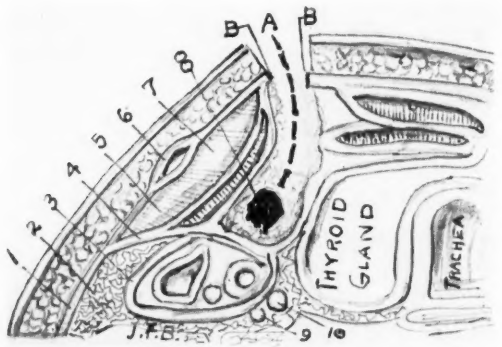

Fig. 5. Shows deep cervical abscess operated upon along line of cleavage. Note how completely all other compartments of the neck are protected from the abscess by the walls of cervical fascia. Note especially that in passing a blunt instrument along this line of cleavage to its bottom all adjoining compartments are walled off and protected against injury. 1—External layer of cervical fascia; 2—connective tissue pocket; 3—carotid sheath; 4—two layers of cervical fascia united after passing around muscle; 5—sternomastoid muscle; 6—external jugular vein; 7—muscle; 8—abscess in the connective tissue pocket. B—Incision through the skin and superficial layer of deep fascia at the anterior margin of the sternomastoid muscle. Note that there is but one layer of fascia at this point and that the muscle compartment is not opened by the incision.

vided excellent surgical advantages. Postgraduate opportunities for advanced study had also been greatly improved, and, as already pointed out, these students had shown tremendous interest in this work.

The writer has been an instructor for many years in postgraduate courses for otolaryngologists and can testify from close contact with these groups that none ever worked with greater zeal in any cause than did these men to prepare themselves adequately. It should require no argument, therefore, to prove that such men are amply qualified. Indeed it may be honestly claimed that some of them are better prepared than those who do general surgery.

Who should practice the "environmental" surgery? Evidently it should be the one who is most capable of doing it. Certainly no one has a just claim unless he has these qualifications, and hence the otolaryngologist should be chosen if better qualified. No one should undertake it without the most thorough preparation, for surgery here presents more difficulties than in other parts of the body.

The wisdom of separating legitimate from environmental surgery is questionable. A surgeon who is capable of operating on a thrombosed dural sinus should be qualified to operate on a thrombosed jugular vein, especially since the dividing line between the two is more or less fanciful. The reason for division of the work is often a local custom and a mere tradition. The general surgeon has always done the environmental work, and hence the belief is common in such places that no one else can do it properly. Members of the general surgical staffs of hospitals would claim such cases and usually would get them; and should the laryngologist perform an operation on a borderline case in such a hospital, he would run the risk of being held up to criticism, especially should the operation turn out badly. This attitude on the part of both profession and laity has greatly retarded the growth of environmental surgery.

In such a city one may see in any hospital on the bulletin of operations for almost any day that, for example, a surgeon will remove an ingrowing toenail at one operating period and will open the neck to evacuate a deep cervical abscess at the next; or he may have done a hysterectomy at one period and followed it immediately by a thyroidectomy. One wonders by what sort of logic one surgeon is permitted to range over the entire body in performing surgery, while another who has had equal and often better preparation is held up to ridicule should he do a thyroidectomy.

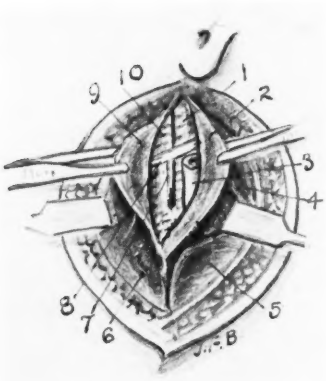

Fig. 6. Showing the external and internal carotid arteries and a portion of the internal jugular vein. An incision has been made through the soft tissues down to the external layer of cervical fascia, as shown in Fig. 1; this latter fascia has then been opened along the interior margin of the sternomastoid muscle and the flaps, including the sternomastoid muscle, have been strongly retracted. The carotid sheath, which was then exposed, was picked up and incised as shown in that figure where the vessels and nerves appear as depicted. 1—External carotid artery; 2—flap of carotid sheath, turned back; 3—internal jugular vein cut off and upper part removed to show the underlying artery; 4 and 5—under side of the sternomastoid muscle, strongly retracted; 6—omohyoid muscle; 7—*descendens hypoglossi* nerve; 8—hypoglossal nerve; 9—flap of carotid sheath; 10—internal carotid artery.

No one should know more about the surgery of the thyroid gland than the laryngologist. Goitre operations should be his special problem. Everything about it suggests that it is his special problem. The gland is bound up as closely to the larynx as were the Siamese twins to each other. The thyroid isthmus crosses the trachea immediately below the larynx. The lateral lobes of the gland rest upon the cartilages of the trachea and when greatly enlarged may dangerously compress it. The recurrent laryngeal nerves enter the larynx after passing near the gland, and palsy of the vocal cords is sometimes attributed to thyroid disease. From a surgical viewpoint, the thyroid is an integral part of the larynx.

Thyroidectomy is easier and usually safer to perform than many operations which the otolaryngologist performs daily with complete success. For example, comparison of certain types of radical mastoid operation with the enucleation of a large cystic goitre will demonstrate that the operation on the thyroid is usually much the easier

of the two. The writer has performed both these operations in large numbers and can truthfully state that enucleation of the thyroid is usually less difficult. Goitre operations are often believed to be more dangerous to life than mastoid surgery, but the danger is frequently due more to the disease than to the operation.

It has been stated that some laryngologists have a fear of operative surgery on the neck. They recall the appalling maze of arteries, veins and nerves which they isolated in their earliest dissections, and they hesitate to face the dangers. They seem not to understand that these dangers may be greatly reduced by one who is willing to devote enough hours of anatomical study under a competent surgical instructor to enable him to gain a complete mastery of the structural relations of the neck*

It should be understood that the pathways leading into the depths of the neck may be made reasonably safe and easy if only sufficient time, energy and proper instruction be devoted to the task.

Subsequent paragraphs of this article state briefly steps that may be taken to insure ease and safety in surgery of the neck.

CONNECTIVE TISSUE AND CERVICAL FASCIA

As previously stated, there are two structures in the neck, a knowledge of which is absolutely essential. These are the layers of fascia which inclose the various structures of the neck and the loose areolar tissue which separates yet binds these structures together. They are usually looked upon as dead and inert and are sometimes cut away by the student in his early dissections, his instructor having no conception of their great value in operative surgery.

THE COMPARTMENTS OF THE NECK

A brief statement of the arrangement of the cervical fascia and areolar tissue and a study of the accompanying drawings should demonstrate the great value of these apparently useless tissues in carrying out easily and safely the steps of almost any operation in the cervical region. Of the two layers of cervical fascia, the outer wraps up all the structures of the neck into one bundle, whereas the inner layer incloses separately each of the three principal compart-

*The author held the chair of surgery of the head and neck in the Indiana University School of Medicine until his retirement. During this period all departmental goitre cases from the several hospitals connected with the Medical School came under his service.

ments. The large blood vessels and vagus nerve fill the compartment of the carotid sheath. The muscles, individually or in groups, fill the muscular compartments, while still another contains the trachea, the esophagus and the thyroid gland. All these divisions of the neck lie one against the other, each in its own container and each filled with a different substance. Each is completely separated from the others.

The separation is made by the layers of fascia, which may be compared to the bulkheads of a ship. Each compartment is bound to those that adjoin it by loose connective tissue. In Fig. 4 it should be noted that the fascia completely separates one compartment from another. The structures inside the carotid sheath have no visible connection with the compartments containing muscles or with the visceral compartment. Note in the illustration that all structures, injury to which would be dangerous, are protected by the strong walls of fascia. It is evident, for example, that an abscess in the compartment containing nothing but areolar tissue (marked 9 in Fig. 4), cannot directly enter the compartment containing the large vessels and nerve unless this wall formed by the fascia is necrosed and broken down. It is further evident that these fascial walls prevent the spread of infection from one to another compartment. But it is the help this fascia gives to the surgeon that the writer wishes to point out and to emphasize. When the separate compartments come into contact with each other, the layers of fascia which surround each are attached loosely together by connective tissue. This arrangement creates a channel that reaches from the surface deeply into the neck, and it is in this channel almost without exception that abscesses and connective tissue tumors form. Hence a natural, safe and easy pathway is provided along which a majority of all surgical affections of the neck may be reached. And here it may again be pointed out that the disease lies in one compartment and the vessels and nerves, injury to which might be fatal, lie in another. Examine Fig. 4 again. Note that the layers of fascia cover the muscular compartment of the sternomastoid muscle, the carotid sheath and the thyroid gland and that this arrangement makes the approach to an abscess or tumor both safe and easy. An incision is made through the skin (Fig. 1) down to the superficial layer of the deep fascia (at 1 in Fig. 2 and at 10 in Fig. 4). This fascia is incised as shown at A in Figs. 4 and 5. The compartment containing the connective tissue and abscess is thus opened. A dull instrument or the finger is introduced through the opening into the compartment and is passed inward, easily separating the loose connective tissue, until the limiting wall of fascia at the bottom is reached. Note again that

muscles, veins, arteries and all other structures are completely walled off and protected from injury by the instrument or finger. These pathways leading to the depths of the neck are spoken of as lines of cleavage.

Since their value is nowhere better shown than in the operation for the removal of a large cystic goitre, it will be helpful to an understanding to give the steps. With the exception of the primary incisions through the skin and the superficial layer of the cervical fascia, which are made with a sharp knife, the greater part of this spectacular operation is done with a dull dissector, usually the index finger. This finger is inserted into the surgical space surrounding the thyroid gland, is swept around the large mass and finally by a dexterous twist of the wrist, flips out the cyst upon the neck. This entire procedure requires but a few moments unless there are adhesions to break up. The speed and safety are possible because of the presence of connective tissue in the surgical space which surrounds the gland and is easily penetrated by the finger.

It seems probable to the writer that when the arrangement of the structures of the neck becomes better known and when the simplicity and safety of treating surgical affections of the fields are better understood, well-trained otolaryngologists will widen their fields to include the surgery of the head and neck. But it should here be pointed out that all operations on the neck are not as simple as just described, for in the event that the region has been seriously inflamed and the tissues have become infiltrated, or in the presence of mestastases from cancer, the lines of cleavage are then obliterated, the walls of the compartments are closely glued together, blunt dissection is no longer possible and sharp instruments must be used. Blind operating is then necessary, and at no time is exact anatomical knowledge more essential than under conditions such as these.

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LXIII

VEGETAL FOREIGN BODIES IN THE BRONCHI: ANALYSIS OF FORTY CASES

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One of the commonest groups of foreign bodies accidentally aspirated in the respiratory tract is that comprising various vegetable substances such as nuts, seeds and fruit pits. Of these the nuts constitute an outstanding majority in the group of cases here reported, peanuts alone making up 90% of the total. Statistics gathered from a group of the country's leading bronchoscopists show the incidence of nuts occurring as bronchial foreign bodies to average 32 of the total. Of late years there has been an ever increasing popularity of confections in which are incorporated various forms of nuts and which are offered for sale, particularly for the delectation of children, in colorful and picturesque forms, with a nomenclature which becomes almost weekly more extensive. Inquiries into the stock of a group of stores reveal the fact that there are at least 12 forms of nut-containing candies in addition to the commoner form of peanuts and other nuts sold in the natural state.

In view of the relative frequency with which a common article of food may become a source of grave danger, particularly to a young child, I feel warranted in presenting an analysis of a group of patients personally treated for aspiration of this type of foreign body. While the total is obviously small in comparison with those seen at some of the larger bronchoscopic clinics, the data have been studied and analyzed with a view to the detailed consideration of the factors which are often omitted in reports of foreign body cases and which are yet of great importance to the general practitioner and the pediatrician who are so likely to be the first to see these cases.

The age incidence in the present series of patients is shown graphically in the chart, Fig. 1. Our youngest patient was ten months old, the oldest (exclusive of the only adult in the group) ten years, the average age being three and a half years. Statistics of all forms of bronchial foreign bodies show a considerable number of patients under one year. Between the ages of six and twelve

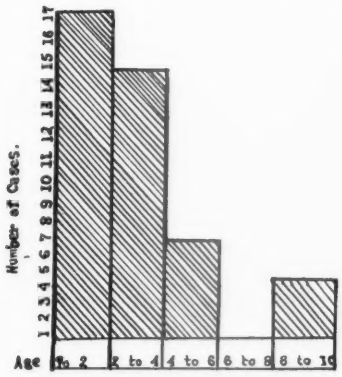


Fig. 1. Chart showing the age incidence in 40 cases of vegetal foreign bodies in the bronchi and the marked preponderance of patients under four years of age.

months, infants making their first acquaintance with their fascinating environment tend frequently to put small objects of every description into their mouths, and by mischance occasionally to aspirate them into the respiratory tract. Vegetable substances, however, during this early period are quite rare, primarily because only through the agency of some older person, a parent or older brother or sister, can the infant secure this form of foreign body. Only with attainment of the more advanced age period of from one to three years, when a more varied diet is in order, do misguided elders allow the baby a nibble of such unfamiliar dietary divergencies as a nut-containing chocolate bar or a few salted peanuts. Induced ofttimes by a crying, laughing or coughing spell, a sudden inspiration draws a fragment or nut into the momentarily unguarded laryngeal orifice, and the damage is done. Older children may of course aspirate a nut, either in pure form or incorporated in candy, on their own responsibility, and paradoxically at times a well-meant parental effort to prevent such a possible accident by sudden admonition has in the very act of eating, provoked a coughing or crying spasm with aspiration which might well have been avoided by permitting normal mastication to proceed unhindered. In any case parents should realize the importance of excluding nuts in all forms from the diet of children under three years of age and preferably in older children. The aspiration of seeds and fruit pits more commonly results from the chance presence of such objects in the mouth as playthings rather than from any intention deliberately to consider them as edible.

Warning should be given in the case of any nut-containing candy as to thorough mastication of the nut particles contained therein.

The securing of a positive history of aspiration of a vegetal foreign body is of the utmost importance, since in these cases one often lacks the positive assistance afforded by the X-ray with opaque foreign bodies. If the event of aspiration, particularly in a child too young to describe the accident accurately, has passed unobserved by any adult, the ensuing signs and symptoms may for a time at least be so mild and insignificant as to be attributed to any one of a dozen less serious and more common conditions. It is therefore the duty of any physician to whom a child is referred for an unexplained cough, a slight but persistent fever, a transient pain in the chest or unusual lassitude or anorexia at least to inquire into a possible history of aspiration. With this end in view he will find himself faced with one of the following situations. 1) The parents will have observed the child to have had a definite choking or coughing spell, usually accompanied by transient cyanosis, coincident with the eating of a vegetable substance, most commonly some form of nut or nut-containing confection. 2) The accident of aspiration may have occurred unbeknownst to the parents, who will therefore deny its existence. 3) The parents may have observed some symptoms suggestive of aspiration to the physician, but unappreciative of their importance will not venture to report them unless their attention is specifically directed to such a consideration. Even a preliminary denial may at first be secured, to be followed, after more mature reflection and a mental review of the events, by an acknowledgment that aspiration may have taken place or that the opportunity for such, at least, existed.

Least of all must the physician who first encounters such a potential foreign body case treat with levity or disregard any involuntary suggestion by the parent or patient that accidental aspiration may have occurred. The assumption, based usually on the absence of outstanding signs of respiratory obstruction, that no foreign body has been aspirated will result all too often in a prolonged sojourn of the offending substance in the bronchus. Failing a positive history, in the presence of signs and symptoms at all suspicious a diagnostic bronchoscopy should be performed. A negative examination with all that it entails is far preferable to an erroneous diagnosis and the development of complications under the guise of chronic bronchitis or unresolved pneumonia.

The aspiration history in the present group of vegetal foreign bodies is summarized in Table I.

TABLE I. HISTORY OF ASPIRATION (40 CASES)

Positive	Negative	Volunteered	Obtained by Secondary Question
38	2	32	6

It thus appears that in by far the greater number of cases parents are aware that some accident has taken place which should merit the immediate attention of a physician. That the parent so frequently does not appreciate the gravity of the situation, as manifested by the elapsed time before many of the patients came to bronchoscopy, is due primarily to the fact that once the offending foreign body has passed the glottis, traversed the trachea and come to lodge in either the right or the left main bronchus, the severity of the initial symptoms subsides and the conclusion is instantly reached that the object has entered the stomach and no harm has been done. This leads to a delay in the summoning of a physician. In Table II is summarized the promptness with which the majority of the patients summoned their local physician and also the extent to which the latter by prompt reference for bronchoscopy, appreciated the importance of the history. In two-thirds of the cases the physician was

TABLE II. PATIENT'S REFERENCE TO A PHYSICIAN

Immediate (24 hours)	Delayed	Prompt Action By M.D.	Delayed Action By M.D.	More Than One M.D.
19	9	16	12	7

called within 24 hours. In 28 of the recorded calls, however, there were 12 instances in which, because of uncertainty of diagnosis, there was a definite delay in reference of the patient for bronchoscopic examination. On seven occasions the patient saw two or more physicians before reaching a hospital. The possible mildness of post-aspiratory symptoms is exemplified in one case in which no physician was consulted until 18 days after known aspiration.

The average time for all patients of three days before reference to a physician in 16 cases becomes of significance when it is compared with the average time interval which elapsed before these patients entered the hospital. For all patients including those who did not consult a physician, this was an average of 12 days. While in numerous instances patients were referred for bronchoscopic treatment within 12 hours of the time of aspiration, this discrepancy

between three days and 12 days indicates the need of stressing the importance of early reference of these patients to a bronchoscopic clinic, at least in those instances in which a positive history of aspiration is given or obtained. In 15 cases in this latter group we again find the time average before reference to a physician to be approximately three days, with a hospital reference time of an average of seven days. This figure of seven clearly indicates the benefits occurring from a knowledge of the history of aspiration as compared with the 12-day average which includes the cases in which no positive history was offered by the parents. The major responsibility rests, therefore, with the parents, first to call a physician in event of anything suggesting aspiration of the foreign body. If this is done, and if the physician then accepts the story at its face value, there will be no appreciable number of cases of delayed removal. Once the question of aspiration has been raised, however, the physician must not let the relative absence of physical signs or mildness of symptoms deter him from seeking bronchoscopic confirmation of the diagnosis or aid in removal. Delay after this point often means increasing difficulty in removal or a likelihood of subsequent complications. In cases of unexplained pulmonary signs, even in the face of a negative foreign body history, bronchoscopy should be resorted to as the final diagnostic measure.

It should be noted that in several of our series of cases, possible aspiration was at first denied, only to be acknowledged on more searching and repeated questioning. A story of aspiration, which would never be given voluntarily, can frequently be obtained if vigorously sought and will rarely prove to be a false scent. A coughing spell, symptomatic so often of other conditions, does not always suggest foreign body to the parents until their attention is specifically directed to the possibility of a half-eaten candy bar, a jar of nuts on the table nearby, or a fall or laugh or cry with something in the mouth. In one of our cases (No. 14) the patient was at the time suffering from whooping cough, and the paroxysm attending inspiration of a peanut might, but for skillful questioning by the physician, easily have passed for a spasm of this common complaint.

ADMISSION SYMPTOMS

We come next to the appearance presented in these known or subsequently proved cases of vegetable substances in the bronchus on admission to the hospital. The essential symptoms are summarized in Table III.

TABLE III. ADMISSION SYMPTOMS

Fever (over 100°)	Elevated Respiratory Rate	Cyanosis	Cough	Hoarseness
16	19	3	29	2

By far the greater number of patients showed no evidence of acute illness, only one case (No. 6) being so classified. While some fever was present in 16 of the 40 cases the average rectal temperature was only slightly over 100°, the highest, 104°, and only five patients showed a fever up to 103°. In these instances of higher temperatures, the average duration of the presence of the foreign body was ten days but there were numerous instances where the object had been known to have been in the bronchus for a far more prolonged time with little or no febrile reaction. This brings up the important question of so-called arachidic bronchitis, a condition characterized by a violent febrile reaction and considered to be due to a peculiar response to certain toxic substances (contained in nuts and certain other vegetal foreign bodies). This feature of these particular foreign bodies has been reported by numerous authors and has been made the subject of an experimental study by Heatly, who showed quite conclusively the irritative effect of certain extracted peanut oils on the lower respiratory tract of animals, which simulated quite closely the symptoms in infants and children who had aspirated nuts, corn kernels, peas, etc. We have been quite surprised and somewhat puzzled in reviewing these cases of aspirated vegetal foreign bodies to find an almost complete lack of this so-called arachidic bronchitis throughout the entire series. Even those cases manifesting a febrile reaction did not fit the clinical picture of the condition as commonly described by other authors. In only one instance (Case 16) did suggestive symptoms appear, but this was after and not before the extraction of the foreign body, in this instance a piece of almond kernel. Incidentally, the almond has the reputation for producing a more than ordinarily virulent form of arachidic bronchitis. In our experience, this same clinical picture has occurred much more frequently as the result of severe streptococcic infections of the upper respiratory tract than of a foreign body, and it is an open question if this infectious element may not be present in certain of the vegetal foreign body cases reported as being the result of some inherent toxic substance in the foreign body itself.

The average pulse rate on admission in the present series of cases was 123, certainly not indicative of any serious toxicity or reaction. In only one instance was a rate of 160 recorded.

Bearing more immediately on the question of bronchial obstruction and its clinical manifestations was the respiratory rate. One thinks naturally of a pronounced rise in this rate as a foregone conclusion in any case in which there is evidence of complete or partial obstruction of the entrance of air into one side of the lung. Nevertheless, the average respiratory admission rate in these 26 patients was only 33, the highest being 50, and yet, as will be shown later, practically all these patients showed bronchial obstruction and were carrying out the oxygenation of their blood with a pulmonary area diminished by from one-half to one-fourth of the normal. The involvement of a similar area in a pneumonia would entail a respiratory rate of from 50 to 70, so that it is quite clear that the obstruction *per se* is not a particularly potent cause of dyspnea. A similar situation is of course observable in patients with an artificial pneumothorax. The rate of 33 is somewhat above an average normal, and 19 of these cases were classified as showing an elevated respiratory rate. Of these only three were classified as truly dyspneic. This factor is stressed, since it is often this lack of signs of urgent dyspnea which leads a physician astray and hence to conclude that, in the absence of more obvious signs of obstruction, the foreign body has been swallowed and not aspirated. Cyanosis, the final stage in respiratory obstruction, was positively recorded in only three instances. Its presence should never be awaited before concluding that bronchial obstruction exists.

Perhaps the one most constant and suggestive sign exhibited by this group of patients was a cough. Even when physical signs were only mildly suggestive and when all other evidences of foreign body failed, a persistent irritative cough was likely to be present. This was so in 29 of these cases. In about half of these the cough was harsh and brassy, in the remainder it was loose and moist. It was more apt to be of the latter type in patients in whom the nut had been present for a considerable period of time and in whom there was beginning to appear a purulent reaction with accumulated bronchial secretions.

Hoarseness was present in only two cases, and in none was the foreign body found engaged in the larynx itself. Once having passed this barrier, there seemed almost no tendency for the foreign body to leave its point of lodgment in either the bronchus or the trachea; hence the absence of any tendency to laryngeal irritation.

PHYSICAL EXAMINATION

Second only to the obtaining of a positive or suspicious history of aspiration are the signs to be elicited by physical examination in cases of nuts or nut fragments in the upper air passages. Within certain limitations dependent upon incompleteness of hospital records, these signs fall into distinct categories and when present go far to establish a positive diagnosis almost equal to X-ray evidence of an opaque foreign body. For this reason, a most careful examination of the lungs is of the greatest importance, together with an accurate interpretation of the findings, since otherwise definite signs may be most misleading and indicate a foreign body on the right side when it is actually on the left. A summary of the essential physical signs is presented in Table IV.

The three commonest signs, so often associated together as almost to constitute a triad, are 1) unilateral limitation of expansion of the lung and chest on the affected side; 2) hyperresonance to percussion (so-called obstructive emphysema); and 3) absent or greatly diminished breath sounds. In the present series of 40 cases, the first of these signs was present 33 times, the second, 30 and the third, 35. All three of them depend primarily on a single physical fact, i. e., a partial obstruction to the ingress of air into the affected lung or part of the lung. Under these conditions, air will enter the lung past the foreign body at the time of inspiration, when the bronchial diameter is somewhat increased. On expiration, however, the bronchial wall contracts, and the foreign body or portion thereof acting in the nature of a valve, prevents the egress of air. As a result, the lung gradually becomes more and more inflated and emphysematous, producing the so-called obstructive emphysema to which attention of late has frequently been called by numerous authors. A lung or its lobe gradually inflated in this manner would produce all three elements of the above-mentioned triad. There will be a limitation of expansion, since such a lung will no longer take part in the normal cycle of inspiration and expiration. Breath sounds will be diminished or absent since after a certain degree of inflation no appreciable amount of air can enter the distended lung. Hyperresonance to percussion will be an obvious sequel to such a situation.

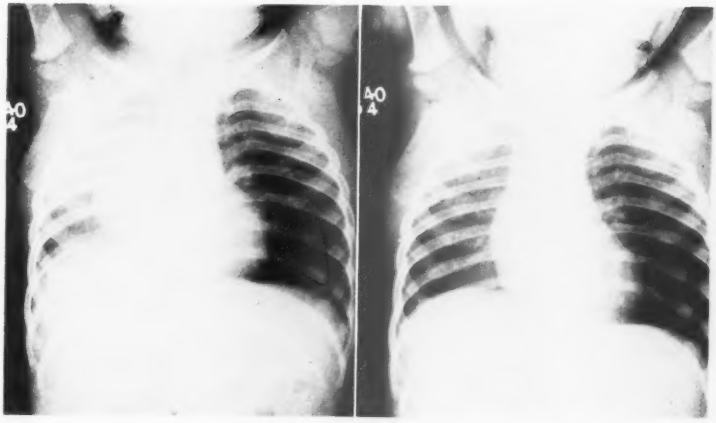
The extent to which signs are present is determined for the most part by the location of the foreign body and the size of that portion of it causing the obstruction. If the lumen of the right main bronchus, for instance, is occluded by a large portion of the foreign body just below the bifurcation of the trachea, then the en-

tire right lung will exhibit the above mentioned signs. If, however, a smaller piece is partially occluding the orifice to the right lower lobe alone, then the area over which these signs are detected will be correspondingly smaller.

The signs just described are those that supervene almost immediately after inhalation of the foreign body. As time goes on and obstruction becomes more and more complete, due to swelling of the bronchial mucosa around the foreign body, the ball-valve action ceases and air can neither enter nor leave the lung. Gradually this air is absorbed, and there then ensues a different picture with the clinical signs of atelectasis. That this is a development requiring more than average time is borne out by the fact that in only six of our patients was it detected, in spite of the fact that several weeks had passed in some of the others who still showed signs of obstructive emphysema. Atelectasis would still manifest the signs of limited expansion and diminished breath sounds, but would differ in the absence of hyperresonance. The sign of dullness or flatness to percussion, recorded in 13 of our 40 cases would of course fit consistently with atelectasis. It appears likewise in certain instances in which there has taken place some exudation of fluid into the lung tissues, following the absorption of the air, constituting what Jackson has termed a "drowned lung". Abscess formation would be the natural outcome of this situation unless the foreign body were removed.

Bronchial breathing appeared only four times and was not due, as other authors have pointed out, to a true pneumonic consolidation. This latter is extremely rare in connection with vegetal foreign bodies, and the signs suggestive of it are far more frequently due to some compression of a lobe adjacent to an area involved in obstructive emphysema.

It is interesting to note that rales were recorded in these cases on only nine occasions. They were indicative of some suppuration, or more frequently of a foreign body loose in the trachea. Here there is no tendency to obstruction of a bronchus, the object being so large as to fail to engage the bronchial orifice. It lies, therefore, loosely in the trachea, often moving back and forth, with coughing, at times audibly striking against the lower surface of the vocal cords and producing an irritative tracheitis. This is accompanied by an excretion of tracheal secretion and the consequent finding of rales. In this situation, both sides of the chest would give essentially negative signs of hyperresonance or diminished breath sounds or limited expansion.



A. *B.*

Fig. 2. Roentgenological appearances in marked obstructive emphysema (Case 35). *A*—Appearance at the end of expiration. The right (normal) lung is deflated and appears distinctly more dense than the left (obstructed lung) which is emphysematous and hyperaerated. *B*—Appearance at the end of inspiration. Both right and left lungs now almost equally inflated, though there is still evident slight emphysema and hyperaeration on the left (obstructed) side. The foreign body in this case was a watermelon seed, not usually thought of as an object likely to act as a ball-valve. It is more nearly comparable to a butterfly-valve, which by rotation on its axis closes the bronchial orifice.

Various combinations of these typical signs may be encountered. There may occur a partial atelectasis in a lower lobe and an obstructive emphysema in a middle lobe, depending entirely on the extensiveness of the obstruction and the location of the foreign body. Particularly must one be on one's guard against misinterpretation of a true compensatory emphysema. This occurs in a normal, unobstructed lung, with some hyperresonance, but with exaggerated, not diminished, breath sounds. It is the result of nature's effort to compensate for the disability of the obstructed lung, which though likewise emphysematous shows diminished expansion and suppressed breathing.

It is interesting to note that there was an equal distribution of the foreign bodies in the right and left bronchus. It is commonly taught that, due to the less angulated junction of the right bronchus with the trachea, objects are more likely to find lodgment on that

side. In the cases here reviewed this dictum does not seem to be substantiated.

X-RAY FINDINGS

While the roentgen-ray examination of cases of non-opaque foreign bodies in the lung must necessarily be far less conclusive than in the case of metallic objects, nevertheless with certain technical refinements and appreciation of detail developed in the past few years, much valuable information can be obtained. Chief among these advances has been the taking of two films, one as nearly as possible at the end of inspiration and one at the end of expiration. This is not always easy or even possible in an uncooperative or crying child, but it affords an opportunity to bring out by X-ray one of the most important diagnostic features exhibited by such bronchial foreign bodies as nuts, that is, obstructive emphysema, to which reference has already been made above in considering the physical signs (Fig. 2). Films of a normal chest taken at the end of inspiration will show equally transparent and slightly hyperaerated lung fields. At the end of expiration, these fields will again be of equal density but definitely less aerated, due of course to the expulsion of all but the residual air from the lungs. If now a vegetal foreign body, acting as a ball-valve, gradually traps air in the obstructed lung with a resultant obstructive emphysema, the roentgen ray picture will be quite characteristic and of distinct diagnostic help. On full inspiration, with air entering both sides of the chest, there will be the normal appearance of bilateral hyperaeration. At the end of expiration, however, a distinct variation is noted. The normal, deflated lung will show a slightly diminished density, as compared with the abnormal or obstructed side, which by reason of imprisoned air will show the same hyperaeration as is seen in both lungs at the end of inspiration. The situation, if not clearly understood, may lead to a totally erroneous opinion as to the location of the foreign body as one would be inclined to regard the more completely aerated lung (at the end of expiration) as the normal one, whereas in reality it is the obstructed one.

If now such changes have occurred in the lung as the result of a more prolonged sojourn of the foreign body in the bronchus, and emphysema has been replaced by atelectasis, then a different picture obtains. At the end of inspiration, only the normal lung is aerated, while at the end of expiration both sides show a diminished radiability, more marked on the affected side. Practically never is any shadow cast by a vegetal foreign body itself visible.

Naturally these findings by X-ray are not always clear-cut, and other factors enter into the picture which must be correlated with the results of the physical examination. The history and physical findings alone would, regardless of the X-ray report, warrant bronchoscopic examination in any case, but confirmatory evidence is always helpful.

Additional information is to be obtained from fluoroscopic examination of the chest. Here a typical obstructive emphysema will show a limitation of motion of the diaphragm on the affected side and a displacement of the heart and mediastinum to the opposite side. Similar limitation of motion without displacement or with displacement to the affected side would be seen in the case of atelectasis.

In those instances where the foreign body is too large to engage in the bronchial orifice it must remain loose in the trachea. In these cases there will, of course, be no X-ray signs of unilateral obstruction, and the roentgenologist cannot be expected to confirm any diagnosis of foreign body.

In the present series under review, there is recorded a positive roentgenological finding of foreign body in 17 cases and a negative one in five. In the latter instance, this does not of course mean that the roentgenologist declared no foreign body to be present but merely that so far as his findings were concerned, they did not tally with the classical signs of bronchial obstruction. In other cases the roentgenologist merely reported his findings without committing himself specifically as to the probable presence of a foreign body. Regardless of this report, other findings in the history and physical examination fully justified bronchoscopy even had no foreign body been found. Such negative X-ray report might be due to the presence of the foreign body in the trachea or to an examination made before any of the classical signs of obstruction had had time to develop.

Obstructive emphysema was clearly noted in 14 cases, and displacement of the heart and mediastinum to the opposite side in the same number. There was unilateral limitation of motion of the diaphragm recorded in 12 instances. Peribronchial infiltration was reported five times and atelectasis of the affected side, eight times, indicative of course of a more prolonged sojourn of the foreign body.

In general the X-ray examination is useful as a part of confirmatory evidence when positive. It is very valuable when it con-

tributes the first suggestion of bronchial obstruction in a case in which, through failure to suspect a foreign body or inquire into the history as to its possibility, this diagnosis had not been previously taken into consideration. Negative X-ray reports, however, have far less bearing on any suspected case of bronchial foreign body.

ANESTHESIA

The question as to what form of anesthesia is best adapted to the operation for removal of bronchial foreign bodies is one which has occasioned considerable discussion among different bronchoscopic schools. Certain operators, feeling somewhat handicapped through infrequent occurrence of these cases and a lack of a trained and ever available team of assistants, prefer the use of a general anesthetic, such as ether, as affording greater relaxation and ease of manipulation and hence as contributory to a greater degree of success in removal. Were there no opposing disadvantages or even dangers in the use of ether, there would undoubtedly be much in favor of this more complete anesthesia, both from the standpoint of comfort to the patient and easier working conditions for the operator. Jackson, however, to whom we must invariably refer as having had a far greater experience in this work than any other living person, has long maintained a position against the use of any anesthetic in removal of bronchial foreign bodies in children and particularly with the type of intruder here under consideration. His reasons rest primarily on the danger of eliminating, by general anesthesia, the action of the accessory muscles of respiration, which in any dyspneic patient are most necessary to maintain the chest expansion under such circumstances. Moreover, should respiration by any chance cease under general anesthesia, it is far more difficult by any artificial means to achieve its resumption than is the case when no anesthesia is employed. While, in our experience, few of these cases of unilateral bronchial obstruction from aspiration of vegetal foreign bodies are in any sense urgently dyspneic, nevertheless, with such possibilities as jamming of the intruder in the trachea or larynx or in the uninvolved bronchus, we are heartily in accord in opposition to general anesthesia so far as ether is concerned. Moreover, ether induces an excessive secretion of mucus, which is an additional handicap in the operative procedure. In the present series 25 of the patients received no anesthetic at all with the exception of a few instances where a small preliminary dose of morphine was administered. On these patients, 17 bronchoscopies were performed. Ether was administered in only three instances.

It must, however, be acknowledged that, for the average operator, to whom these patients come with some irregularity and infrequency but who must because of his location nevertheless take care of them when they do, the difficulties of working on a struggling child in the performance of this delicate procedure present no small problem. True, he should have available capable assistants, trained in the restraining of such patients, and he should, obviously, maintain his own skill at the highest possible level by regular practice in the laboratory or on the mannequin. Neither of these forms of practice, however, duplicates the situation encountered in the operating room, and if, in spite of his best efforts, the operator finds himself handicapped by the varied factors incident to working without anesthesia, he cannot but wish that there were some form of anesthesia to which he might have recourse, provided always that it adds not the slightest risk to the procedure. In addition, were any such anesthetic to be found, it cannot be denied that the sparing to the patient of an obviously uncomfortable, if not actually painful, ordeal would be an added advantage. Any form of morphine in doses sufficient to produce appreciable analgesia would be quite unsafe because of the respiratory depression and is mentioned only to be condemned. Is anything else worthy of consideration? During the past five years we have employed the rectal anesthetic, avertin, in several bronchoscopic examinations of cases of chronic pulmonary suppuration. We have been struck with the manner in which the procedure was facilitated, with the complete analgesia afforded the patient and with the satisfactory manner in which the protective laryngeal and cough reflexes were maintained. The patient's circulatory and respiratory functions were in no way embarrassed or depressed and we felt that the use of such an anesthetic might have some place in foreign body removal for certain operators and under certain conditions. It would, of course, be out of the question for any patient showing the slightest signs of dyspnea or circulatory embarrassment and in any instance in which the patient's condition suggested that its use would contribute the least additional risk. In many other instances of bronchial obstruction, such as that occasioned by the foreign bodies here under consideration, we feel that it may, when used judgmatically, contribute to more successful removal than might otherwise be possible. It must be given by an anesthetist skilled in the administration and with the careful observation of all the details which must be carried out.

In the present series of nuts removed from the respiratory tract, avertin by rectum was used in 13 bronchoscopies on 11 patients.

The dose varied from 80 to 100 mg. per kilo of body weight. That its use lessens the strain on both operator and patient cannot be denied, though it may still be criticized with some validity. In only one instance was there any complication which could be construed as the direct result of the use of avertin. In another case there was a brief period following removal of a portion of peanut in which a slight acapnia developed, which was easily combated with the inhalation of carbon dioxide. A summary of the complications in this series of cases, to be taken up in detail later, shows no preponderance of trouble occurring in patients anesthetized with avertin over those without any anesthesia. The only fatality occurred in a case in which no anesthetic was given.

For the operator who is seeing these cases with reasonable frequency, who has a trained team of assistants always available, and whose technique is quite able to cope with the unavoidable resistance of a conscious patient, the use of any anesthetic is quite unnecessary. For those operators who do not work under these desirable and fortunate conditions, avertain would seem to offer certain advantages if used under properly controlled conditions.

OPERATIVE AND BRONCHOSCOPIC FINDINGS

The operative technique involved in the removal of the foreign bodies here analyzed has many attributes common to any bronchoscopic procedure and hence will be dealt with here only in considering such features as are peculiar to nuts. Among these features are to be mentioned, first, localized reaction to the foreign body, second, technical difficulties in removal and, third, immediate postoperative reactions.

As mentioned above, many authors have called attention to the unusual reaction occasioned by the presence of nuts and other vegetal substances lodged in the respiratory tract. This has been manifested by intratracheal and bronchial inflammation and a high toxic febrile reaction. Strangely enough, none of our patients exhibited this reaction, even when the foreign body had remained *in situ* for periods of time up to five weeks. Whether this fact is in any way related to differences in the state of the foreign body when aspirated, that is cooked or uncooked, is difficult to say. Of the fact that such reactions have been frequently observed, there can be no doubt. In our own cases, the reactions set up by the invader were recorded as negative in 13 instances and as consisting in seven cases of such an edema and inflammation of the bronchial wall just proximal to the foreign body as partially to obscure it. Definitely purulent secretions were

encountered only six times, and pure blood and mucus were each mentioned once. It is interesting to inquire into the relation between these findings and the length of time that the vegetal substance had been present in the respiratory tract. With a few exceptions, the marked signs of local inflammation as evidenced by edema of the bronchial wall or by purulent secretions occurred in those cases in which the foreign body had been present for three weeks or more. This is, of course, what might be expected and is an obvious point in support of the contention that the earlier the diagnosis is made the easier will be the problem of removal. An annular ring of edema, obscuring a large part or whole of a nut fragment, adds considerably to the difficulty of removal, as does also the presence of purulent secretions which require repeated aspiration before removal may be safely attempted.

Technical Difficulties. Foreign bodies such as nuts do not, of course, present the problem so often encountered in dealing with pointed objects such as nails and tacks, and removal *per se* is not likely to do any damage to the bronchial wall. What may best be termed fragmentation is perhaps the greatest obstacle encountered in the removal of nuts. There is a tendency for such objects to break by cracking when grasped with forceps, and the longer their presence in the bronchus and their contact with bronchial secretions, the greater this tendency to fragmentation. Attempts to grasp the object delicately may mean an insecure hold, while more forcible application of forceps may break a large nut fragment into several smaller ones. The dangers of this situation are quite obvious. If the portion held by the forceps is retained and removed, there is the possibility that smaller pieces may yet remain behind unknown to the operator unless subsequent symptoms indicate that removal was not complete. This is the more likely since the smaller fragments may easily descend to a lower position in the bronchial tract, at a level too low to permit visualization through the bronchoscope. Occasionally such smaller fragments may be coughed up spontaneously, but one can hardly rely on this fortunate accident with any confidence. What is far more hazardous is the possibility that a relatively large fragment may be broken off and aspirated into the previously unobstructed bronchus, in which case, if the originally affected side be in any degree occluded by inflammatory reaction, there will occur a bilateral obstruction with asphyxia, calling for immediate removal of the loose secondary fragment, if the fatality is to be avoided. Such was the situation in one instance (Case 13), where only prompt retrieving of the fragment of a peanut from the

opposite lung saved the patient from asphyxiation. This unpleasant situation is most likely to arise when the original foreign body is in the trachea and so large as to prevent its entrance into either bronchus. If fragmentation then ensues in the process of removal, smaller but still good-sized pieces may then easily occlude both bronchi almost simultaneously.

To check up on this situation and to avoid so far as possible the leaving of unremoved foreign bodies in the lung, we have made it a practice to auscultate the lungs before the termination of bronchoscopy. If the quality of the breath sounds in any way suggests gross failure of air to enter one side of the chest, further search is immediately made for another portion of the foreign body. This plan is based on the fact that in most instances, except after prolonged sojourn, an occluded lung will resume its function almost immediately after removal of the obstructing foreign body, at least to such an extent as to make further gross obstruction quite obvious with the stethoscope. In this way, it may be possible to avoid the necessity of a secondary bronchoscopy at some later date.

A review of our cases reveals the fact that in 20 instances the foreign body was removed in a single unit and in 15, as two or more fragments. In this connection, like most operators, we have had bizarre experiences in which a fragment detached from the original object has been coughed up through the bronchoscope, or impinging against the end of the tube, has been extracted with it, but such events are far more a matter of good luck than good management and should not be relied upon to supplant a properly executed method of forceps removal. An interesting technique of foreign body removal by aspiration was advocated by Dr. Campbell Smyth and would appear to have at times certain distinct advantages over the use of forceps.

In the few instances in which the foreign body lay loosely in the trachea, some modifications of technique were necessary. Here the risk of the object's stripping off the forceps at the narrower subglottic space is greater, and the need for a firm hold is all the more important. Were the foreign bodies to become jammed at this point, serious respiratory obstruction would inevitably result. It would then be necessary to return the foreign body to a lower level and reapply the forceps more firmly but with the ever-present risk of breaking it into small pieces.

Multiple bronchoscopic examinations were necessary in seven of our cases. In two of them this was the result of failure at the first

or second examination to locate any foreign body. It is surprising how such an examination, though carefully carried out, may at times fail to reveal a foreign body which on a subsequent bronchoscopy is at once quite obvious. Some slight change in position or alteration in local reaction usually accounts for this. In any event, it is a safe conclusion that, where history or physical findings strongly point to the presence of a foreign body, failure to observe it bronchoscopically on one or even two examinations does not guarantee its absence, and further search should be made. So long as these examinations are reasonably spaced, such multiple bronchoscopies do no harm and are quite likely in the end to yield success.

The same may be said of multiple bronchoscopies in which removal of remaining fragments is deliberately delayed till a later date. It may be far more advisable to terminate the operation with this end in view, provided the patient's condition is satisfactory, than to prolong the first examination unduly.

Our series shows three instances in which this plan was carried out with ultimate success. Fragments were deliberately allowed to remain with the idea of removing them at a second sitting. Since these fragments would of necessity be smaller than the original, it is quite unlikely that they would give rise to any serious, sudden obstruction. Naturally, the more extensive one's experience with this type of case, the less frequent would be the necessity of secondary removal of fragments. In one instance (Case 16) five postoperative bronchoscopies were required to deal with tracheal bronchial crusting, a condition to be considered under the heading of complications.

COMPLICATIONS

Theoretically the removal of a vegetal foreign body in its entirety from the trachea or bronchus should not be followed by any complications attributable to the operative procedure itself. Such an achievement must depend primarily on the skill of the operator and the extent to which any trauma is avoided. Apart from this aspect, it is quite obvious that in certain instances when the foreign body has been present for an undue length of time, or when it has induced a high degree of local reaction, there will be present, even after rapid and skillful operative removal, certain abnormal physical findings which, however, cannot be properly classified as complications. Residual suppuration in a lung or localized inflammation may not infrequently require time for its subsidence, and the patient may, during this period, exhibit evidence of pulmonary involvement

in spite of anything that the operator can do and for which he is in no way to blame.

Such conditions as laryngeal edema, evidence of residual fragments, or unexplainable febrile reaction with or without signs of pulmonary involvement not previously present, must be regarded as true complications and hence indicative of a situation which requires explanation and perhaps additional therapeutic measures.

Viewed in this light, the present series of cases here analyzed present certain interesting facts. In 12 instances there was absolutely no postoperative symptomatology, indicating that the preoperative effects of the foreign body were so slight as to produce no residual signs and that the postoperative reaction itself was nil. This is the ideal type of case, with a result dependent on an early diagnosis, prompt and rapid removal, and the fortunate absence of any violent reaction on the part of the respiratory tract to the foreign body itself.

In 13 cases, there is a record of some symptomatology, such as fever, brassy cough, purulent expectoration or physical signs of pulmonary involvement, all of which however, subsided or disappeared spontaneously without requiring any further bronchoscopic or surgical procedure. In all of these, one must consider the findings as due primarily to the marked effects of the presence of the vegetal substance rather than as complications of its removal. It is quite possible that in certain instances, the operation of removal may have temporarily aggravated these symptoms, but in view of their gradual subsidence, they may fairly be excluded from the group of truly operative complications. Nevertheless, all such signs and symptoms must be carefully watched and interpreted so long as they are present, and in the event that suspicion is aroused at any time that matters are not proceeding satisfactorily, further bronchoscopic examination might be advisable primarily on the suspicion that a small fragment still remained in the lung. To allow this situation to go unrecognized and untreated would, of couse, be to subject the patient to still more prolonged lung suppuration. The length of time before one would intervene secondarily for such a situation would, of course, depend on the degree of pathology present at the time of the original bronchoscopy. The greater the initial involvement of the lung, the longer would be the expected period of convalescence following removal of the foreign body. Frequent checking by physical examination and X-ray unit will usually indicate whether satisfactory progress is being made.

In 11 cases there is a record of symptoms plainly indicative of an unremoved fragment of foreign body. In three of these, there was a prompt subsidence of these symptoms following secondary removal of the remnant, and in a fourth, the patient coughed up the remnant spontaneously. Fragmentation has already been discussed, and it only remains here to reiterate that such an accident must always be borne in mind as a possibility and as an explanation for signs and symptoms which do not satisfactorily clear up. It is an accident which will occur less frequently as the operator becomes increasingly skillful in the work but one which the average bronchoscopist must expect to deal with from time to time. So long as the situation is recognized in time, a secondary removal will do no harm and will usually be followed by a prompt subsidence of the residual signs which furnished its indication.

In Case 2, three bronchoscopies were required for successful removal because at the first two examinations no foreign body could be seen. Such a negative examination, while disappointing, is not to be deplored, provided it adds no further traumatic disturbance to the airways. It by no means rules out the presence of the foreign body, if the suspicious signs and symptoms persist unchanged. "Try again" is a good bronchoscopic motto, when a foreign body is strongly suspected and not found at the first or even at the second examination.

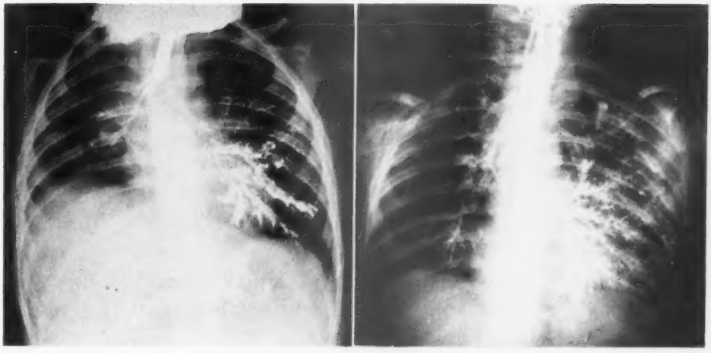
In four instances, it was necessary to carry out further procedures for postoperative laryngeal obstruction. In one of these (Case 16) there was no obvious explanation for the development of the complication, since it followed a short bronchoscopy for removal of an almond fragment of brief sojourn. Since the condition was considered to be one of purely postoperative laryngeal edema, direct intubation was carried out with only temporary relief. Tracheotomy was next resorted to, following which there developed typical signs of low tracheobronchial infection, with the production of inspissated crusted secretions which required bronchoscopic removal on four occasions. The picture at this time was typical of what we have elsewhere described as acute laryngotracheobronchitis. Fortunately the severity of the symptoms gradually abated in a few days, but it was four weeks before decannulation could be accomplished. Though none of these complicating signs was present before bronchoscopy, it is possible that this case was one of so-called arachidic bronchitis, a possibility made more likely by the fact that the almond is said to be particularly likely to cause such a severe reaction. Three other tracheotomies were necessary to deal with transient postoperative

laryngeal obstruction. Such laryngeal edema is relieved entirely by intubation, but if extubation cannot be achieved after two trials it is wiser to resort to tracheotomy. Such a complication may be attributable to the operator's lack of skill in removing the entire foreign body at the first sitting, though it may sometimes be best to discontinue the bronchoscopy after a first portion has been extracted.

The possibility of permanent damage to the respiratory tract following foreign body aspiration must always be considered. Many experienced bronchoscopists have repeatedly asserted that most pulmonary suppuration incident to even a prolonged sojourn of a foreign body will undergo resolution following removal of the intruder. Unquestionably this is true particularly with respect to metallic foreign bodies. In the present series there is only one instance (Case 27) in which it is believed that permanent pulmonary damage followed the removal of a prune stone present in the bronchus for at least one year. In this case no aspiration history was obtained until misdirected treatment had induced a phrenicectomy. Though the patient is essentially asymptomatic, roentgen examination of the chest still reveals opacity and evidence of fibrosis.

A more unusual situation is that of Case 40, in which there was clean-cut evidence, after removal of a peanut, of what is believed to be temporary bronchiectasis (Fig. 3). It is not generally believed that spontaneous recovery with restitution of a normal bronchial tree ever occurs after true bronchiectasis. A subsequent film (B), however, certainly gives the impression that the original bronchiectatic dilatations have disappeared, and symptomatically there is nothing to suggest the existence of a permanent bronchiectasis. It is possible that more extensive roentgenological investigation might show bronchiectasis to be a more common sequel of vegetal foreign bodies in the bronchi than is now thought to be the case. Certainly none of the follow-up examinations in the present series would lead to the suspicion of its existence in any of the patients.

We had hoped to be able to report this small series of cases of vegetal foreign bodies in the respiratory tract without having to record a fatality, but the otherwise unbroken run of successful results was marred by one case, No. 6. This patient had previously been treated elsewhere as a case of pneumonia, and only after further scrutiny into the history five weeks later was the suspicion of inhalation of a peanut elicited. Bronchoscopy without anesthesia was performed, and an obvious nut fragment was seen occluding the right main bronchus. This was grasped and removed, but the por-



A. *B.*

Fig. 3. *A*—Showing typical appearance of left-sided bronchiectasis after lipiodol injection, two months after aspiration of a peanut. *B*—Appearance in same patient four months after aspiration, with restitution of almost normal bronchial delineation.

tion recovered seemed smaller than that seen through the bronchoscope. As further search was being made for the remainder, the child suddenly ceased to breathe, and, still more alarming, no heart sounds could be heard. In spite of all the classical methods of resuscitation, no signs of life were evident from the moment of suspended respirations. Immediate search of both bronchi failed to reveal any further peanut fragments, and since autopsy was refused, it was never possible to determine the cause of death. As a result of a prolonged illness, the patient was undoubtedly in a greatly debilitated state, and it would perhaps have been wiser to have postponed bronchoscopy until some such supportive measure as a transfusion or intravenous glucose administration might have aided in raising the preoperative resistance. The suspicion that the entire nut fragment was not removed still leaves open the possibility that the remaining portion entered the previously unobstructed bronchus though the signs were much more those of cardiac collapse than of bilateral bronchial obstruction.

SUMMARY

Forty cases are reported of vegetal foreign bodies aspirated into the respiratory tract and removed by bronchoscopy. The importance is stressed of inquiring into the possibility of aspiration in taking the

history of any case of unexplained pulmonary pathology. The characteristic physical findings are summarized and shown in the majority of instances to be quite typical in this condition. X-ray reports are summarized and the typical findings illustrated and variations explained.

The question of anesthesia is discussed, and the merits of no anesthesia, avertin per rectum and ether are considered.

Bronchoscopic findings are analyzed, and the problem of fragmentation of the nuts is dealt with in detail.

Complications are considered and estimated according to the immediate or later effects, together with such measures as tracheotomy or intubation, necessary to combat them.

1. The majority of the patients reviewed above were treated at the Children's Hospital in Boston. The remainder were seen at the Peter Bent Brigham Hospital and at the Bronchoscopic Clinic of the Massachusetts Eye and Ear Infirmary of Boston.

2. Acknowledgment is made to the New England Journal of Medicine for permission to utilize and reprint certain portions of the author's article, "Nuts as Foreign Bodies", printed in the Journal on October 11, 1934.

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TABLE IV.—PHYSICAL EXAMINATION

Case	Consolidations (Atelectasis or Pneumonia)	Bronchial Breathing	Dullness or Flatness to Percussion	Unilateral Limitation of Expansion	Hyperresonance or Emphysema	Absent or Markedly Diminished Breath Sounds	Rales
1.	0			+(L)	+	+	0
2.	0			+(L)	+	+	0
3.	0			+(L)	+	+	0
4.	+		+	+(R)	0	+	0
5.	0	+	+(R)	+(R)	0	0	+
6.	+(R)	+(L&R)	+(R)	+(R)	+(L)	+(R)	0
7.	0	0	0	0	+(R)	+(R)	+
8.	0			+(L)	+	+(L)	0
9.	+	+	+	+(R)	0	0	0
10.	0	0	0	+(R)	+	+	0
11.	+(LL)		+(LL)	+(L)	+(LU)	+	0
12.	+(LU)		+(LU)	+(L)	+(R)	+(L)	0
13.	0	0	0	0	+(L&R)	+(L&R)	+
14.	0		+(R)	+(R)	0	+(R)	0
15.	+(RL)	0		+(R)	0	+(R)	0
16.	0	0	0	+(L)	+(L)	+(L)	0
17.	0	0	0	+(L)	+(L)	+(L)	0
18.	0	0	0	0	0	0	0
19.	0	0	0	+(L)	+(L&R)	+(L)	0
20.	0	0	+(LL)	+(L)	+(LU)	+(L)	0
21.	0	0	?	+(L)	+(L)	+(L)	0
22.	0	0	0	+(L)	+(L)	+(L)	0
23.	0	0	+(RL)	+(R)	+(L,RU)	+(R)	0
24.	0	0	0	0	+(L)	+(L)	0
25.	0	0	0	+(R)	+(R)	+(R)	0
26.	0	0	0	0	+(R&L)	+(R&L)	+
27.	0	0	+(R&L)	+(R)	+(RU)		+
28.	0	0	+(R)	+(R)	0	+(R)	+
29.	0	0	0	+(R)	+(R)	+(R)	0
30.	0	0	0	0	0	+(R)	0
31.	0	0	0	+(L)	0	?	0
32.	0	0	0	+(R)	+(R)	+(R)	0
33.	0	+(RU)	+(RL)	+(R)	0	+(R)	+
34.	0	0	0	+(R)	+(R)	+(R)	0
35.	0	0	0	+(L)	+(L)	+(L)	0
36.	0	0	0	+(R)	+(R)	+(R)	0
37.	0	0	0	+(L)	+(L)	+(L)	0
38.	0	0	0	+(R)	+(R)	+(R)	0
39.	0	0	+(L)	+(L)	+(R)	+(L)	+
40.	0	0	0	+(L)	+(L)	+(L)	0

R—right; L—left; LL—left lower; RU—right upper; RL—right lower; LU—left lower.

SUMMARY OF PHYSICAL SIGNS

Right-sided involvement	in 17 out of 40 cases
Left-sided involvement	in 17 out of 40 cases
Side involved not discernible	in 6 out of 40 cases
Unilateral limitation of expansion	in 33 out of 40 cases
Hyperresonance (emphysema)	in 30 out of 40 cases
Absent or diminished breath sounds	in 35 out of 40 cases
Rales	in 8 out of 40 cases
Consolidated or atelectasis	in 6 out of 40 cases
Bronchial breathing	in 4 out of 40 cases
Dullness or flatness to percussion	in 13 out of 40 cases

TABLE V.—TABULATION OF 40 CASES OF VEGETAL FOREIGN BODIES IN THE BRONCHI

Case No.	Age (Yrs.)	Duration of Sojourn	Foreign Body	Anesthesia	No. of Bronchoscopies	Complications	End Results
1.	1	4 days	Peanut	None	1	None	Cured
2.	2	20 days	Peanut	Avertin, Ether	3	None	Cured
3.	1¼	14 days	Peanut	Avertin	1	None	Cured
4.	1	7 days	Peanut	Avertin	1	Rt. pneumonia	Cured
5.	2¾	4 days	Peanut	None	1	None	Cured
6.	3½	22 days	Peanut	None	1	Respiratory failure	Died
7.	8	3 hours	Peanut	Avertin	1	Retained fragment	Cured
8.	2	2 days	Brazil nut	None	1	None	Cured
9.	3	5 hours	Peanut	None	1	None	Cured
10.	1⅓	3 days	Peanut	None	1	Fragmentation	Cured
11.	5	9 days	Peanut	Avertin	1	None	Cured
12.	2	?	Peanut	None	2	None	Cured
13.	3	6 days	Peanut	None	2	Fragmentation	Cured
14.	3	4 hours	Peanut	Avertin	1	Temporary apnea	Cured
15.	5½	6 weeks	Peanut	None	2	Fragmentation Intubation	Cured
16.	20 mo.	5 hours	Almond	Avertin	5	Tracheotomy Tracheobronchitis	Cured
17.	2½	11 days	Peanut	Avertin	1	None Intubation	Cured
18.	1½	5 weeks	Peanut	None	1	Tracheotomy Fragmentation	Cured
19.	2	4 weeks	Peanut	Avertin	1	None	Cured
20.	2¾	3 weeks	Peanut	None	1	None	Cured

TABLE V (CONTINUED)

CASE NO.	AGE (Yrs.)	DURATION OF SOJOURN	FOREIGN BODY	ANESTHESIA	NO. OF BRONCHOSCOPIES	COMPLICATIONS	END RESULTS
21.	4	9 days	Peanut	None	1	None	Cured
22.	11	3 days	Peanut	None	1	None	Cured
23.	5	14 days	Peanut	None	1	None	Cured
24.	4	10 days	Peanut	Avertin	1	None	Cured
25.	17 mo.	4 weeks	Peanut	Avertin	1	None	Cured
26.	2⅓	1 day	Peanut	None	1	P. O. fever	Cured
27.	10	12 mo.	Prune stone	Avertin	1	None Tracheotomy	Residual bronchiectasis
28.	2½	24 hours	Kernel of corn	None	2	Tracheobronchitis	Cured
29.	4	5 weeks	Cucumber seed	None	1	None	Cured
30.	2½	16 days	Peanut	None	1	None	Cured
31.	2½	Several weeks	Apple skin	None	1	None	Cured
32.	2	4 weeks	Peanut	None	1	None	Cured
33.	49	3 months	Peanut	Local	1	None	Cured
34.	3	1 week	Peanut	Avertin, Ether	1	None	Cured
35.	10 mo.	12 hours	Watermelon seed	Ether	1	None	Cured
36.	2	11 days	Peanut	None	1	None	Cured
37.	15 mo.	18 days	Peanut	None	1	None	Cured
38.	2	?	Peanut shuck	None	1	None	Cured
39.	5½	4 weeks	Peanut	None	1	Fragmentation Intubation	Cured
40.	15 mo.	3 days	Peanut	1 Ether 2 None	3	Tracheotomy Bronchiectasis	Cured

LXIV

PRACTICAL TECHNIQUE FOR TRACHEOTOMY*

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True emergencies, in otolaryngology, are rather infrequent and tend to decrease as the general standard of medical practice improves. With better diagnosticians being developed by our medical schools and with increased interest in continuation education throughout the country, earlier and more accurate diagnoses are being made, resulting in the more frequent reference of patients before conditions progress to the point of emergencies. Laryngeal obstruction, however, still remains an emergency—probably the outstanding emergency, not even excepting hemorrhage, calling for the services of the trained laryngologist. This may develop so rapidly, or its onset be so overlooked, as to make necessary a tracheotomy under very unfavorable conditions. In my own experience this is about the only operation that may have to be performed any time and anywhere, perhaps in some distant community hospital or even in a private home. With rare exceptions the general practitioner still stands helpless in this emergency. Too frequently his call for assistance is belated.

The facts just stated are the reasons for this paper. It will attempt to describe certain modifications in the technique of tracheotomy which have been found especially helpful under what are sometimes trying conditions. For while in the usual textbook description, tracheotomy is a relatively simple procedure, anyone who has been obliged to perform this operation on the thick neck of a fat infant, struggling for breath, on an improvised operating table with inadequate light and complete lack of trained assistants, will agree it is not always either simple or easy.

For the extreme emergency, when the physician is faced with the necessity of relieving laryngeal obstruction without adequate instrumentation or preparation, the procedure described by Jackson[1] meets all requirements and should be taught to all practitioners of medicine. But these occasions, on the highways and byways of life,

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as it were, when the physician is called upon to play a heroic role are, fortunately, rare.

For the orderly tracheotomy, when there is no immediate fear of laryngeal obstruction, or when performed as a preliminary measure to some laryngeal operation, the usual classical procedure suffices.

The majority of cases requiring tracheotomy will be found in that group with beginning laryngeal obstruction and, while not necessarily in extremis, they must be considered as emergencies. There is no necessity for neglecting ordinary surgical principles, but there is no time to waste.

The type of patient that this paper is especially concerned with will be operated upon perhaps in a small community hospital, without assistants trained in the procedure, and may have to be left in the care of local physicians and nurses who, quite possibly, may never have even seen a tracheotomy before. It is rather appalling to think of the number of internes and nurses who leave our hospitals without any experience of this kind whatsoever. If we are lucky there will be suction available.

None of the modifications suggested are original. They all have been reported before this Association at various times. I have simply adopted these ideas, combining them into a routine which has proven quite satisfactory.

Briefly the purpose in handling these cases is, first, to convert the emergency into an orderly procedure by eliminating the immediate danger of asphyxia; and, second, to allow for a safer and easier aftercare at the hands of inexperienced personnel. In 1936, before this Association, Dr. Chamberlain[2] strongly advocated the use of the Mosher life saver tubes for intubation of the trachea preliminary to tracheotomy. Valuable as these are, in my opinion they are second choice to the bronchoscope introduced under direct vision. This is an old procedure, practiced by many laryngologists, yet usually mentioned only sketchily, if at all, in most textbooks. The history obtained in these cases is not always reliable, and there is the possibility that the obstruction may be due to a laryngeal foreign body. Under such circumstances the advantages of direct inspection are obvious. Another decided advantage is the possibility of keeping the lumen clear by the use of a suction cannula during the time before the trachea is opened.

It is interesting to note that the original tube designed by Dr. Mosher was actually a straight tracheoscope, to be introduced by means of the laryngeal speculum. This had distal illumination, a decided advantage, and an obturator, occasionally useful. Later the tube was modified[1] to its present form at the suggestion of Dr. Fink, to allow introduction by the indirect method. While the time element may indicate the use of the indirect tube, the advantages of a distally illuminated tube, open for suction, such as the bronchoscope, make it the instrument of choice whenever possible. It only takes a few seconds longer to insert, provided the instruments are ready and at hand.

A small bronchoscope, suction cannula and laryngeal speculum are just as much a part of our routine set-up for tracheotomy as are the usual instruments. Battery and cords go with this set when called to some outside hospital. The bronchoscope, already sterile and with lamp fitted, can be quickly introduced and then held in place by a nurse, while the operator scrubs up and the neck is prepared. Should the lumen become clogged it is easy to clear it with the suction cannula, an advantage over the indirect tube.

The operation is then carried out in the customary manner, so beautifully described by Jackson, until we come to opening the trachea, with emphasis upon adequate incision, hemostasis and the avoidance of undue denuding of the trachea. Bleeding will have been decreased as the preliminary intubation will have relieved the vascular congestion in the pretrachial veins, resulting from the laryngeal obstruction.

Some years ago Mosher originated, but never published, a procedure which he called "punch tracheotomy." I am inclined to think that a better designation would have been window or open tracheotomy, as the word "punch" has a little too much suggestion of a rough stabbing operation, which this procedure certainly was not. The idea of cutting a window in the tracheal wall apparently originated many years ago in Europe. In Richards'[3] excellent article he gives credit for this first to Dieffenback and states that later Hall suggested the procedure in order to avoid the use of a cannula. St. Clair Thomson,[4] in his textbook, advocated cutting away the edges of the tracheal ring with scissors. In Dr. Mosher's operation, after exposure of the trachea, the thyroid isthmus is cut, clamped and each side carefully sutured. Emphasis is placed upon complete hemostasis. Neglect of this may allow bleeding into the trachea. Then a crucial incision is made in the trachea, following which the

four triangular flaps are removed, leaving a round opening in the tracheal wall. Dr. Mosher removed these flaps with a tonsil punch-forcep, hence the name punch tracheotomy. The stumps of the thyroid isthmus are then sutured to the ribbon muscles and they in turn to the margins of the skin incision, keeping the edges separated and bringing the tracheal opening to the surface. If correctly performed there is no need for a tracheotomy tube for the first week. Later the closing in of the muscles makes a tube necessary. Quoting Dr. Mosher,[5] apropos of this operation, "After an open tracheotomy the first night is a quiet one for patient, nurse and doctor. We all know how tempestuous it often is if a tube has been placed in the trachea. It has the great advantage that drainage in a child can be obtained by inverting the patient. In peanut bronchitis where the lungs fill rapidly with watery secretion the inversion method of drainage is especially useful."

While this procedure was designed primarily for use in foreign body cases with fluid in the lungs, it has been found extremely useful in the type of cases under consideration. The original technique may be modified somewhat according to circumstances, but the general idea is pretty much adhered to. This allows one to leave the patient in rather inexperienced hands with more of a feeling of security than would otherwise be possible.

All bleeding points are clamped and carefully tied. The stumps of the thyroid isthmus are very carefully sutured, not merely tied, and are then sutured to the ribbon muscles. The bronchoscope has been left in place, so that the use of hooks to anchor the trachea is unnecessary. Incision in the mid-line over the tube obviates the danger of misplaced or too deep incision. The edges of the flap are picked up with rat-tooth forceps and excised with scissors. The bronchoscope is now withdrawn and the trachea cleaned of any secretion by the suction cannula. Then a properly fitting Jackson tracheotomy tube is inserted. This procedure is not performed with the idea of doing away with the tube but to facilitate changing afterwards, and to provide a safeguard should the tube come out. An exact duplicate of the tube is always provided and the physician instructed to change it daily. Replacement of the tube for the first few days after tracheotomy has not always been easy or devoid of danger in untrained hands, with the longitudinal incision, and there would be a tendency to leave well enough alone, letting the tube remain for days without cleaning. This method does away with that difficulty, making replacement quite simple. Sometimes it has not

been necessary to divide the thyroid isthmus, there being ample exposure below, so that the tracheal window was easily accessible, but, as a rule, it seems better to do this. Certainly this "exteriorizing" of the tracheal opening facilitates the subsequent care of the patient, and the operator feels less uneasy when circumstances make it impossible for him to supervise at least the postoperative period. With instructions to the local physician and nurses as to the use of a suction catheter, keeping the airway clear, providing moisture and fluids, etc., these cases do very well, unless bronchoscopic removal of crusts becomes necessary. In such a contingency there is no substitute for the constant presence of one trained in bronchoscopy.

There seem to be no untoward results from this procedure. The patients heal nicely after decannulization and with little scarring. I have not seen any cases of stenosis resulting from this, and I understand that neither has Dr. Mosher with his far greater experience.

Richards[3] has shown by experimental work that there is little tendency for encroachment on the tracheal lumen from a large defect in the anterior wall. He advocates a crucial incision, feeling that stenosis is far less likely to develop from this than from the single longitudinal incision, where pressure of the cannula may depress the cut edges of cartilage. This is likewise avoided by the oval window which, in addition, has the advantage of easier changing of the tube in inexperienced hands.

SUMMARY

As I said in the beginning there is nothing new or original in this procedure. I have called this a practical technique because it works the best in my hands, under what are sometimes difficult circumstances. The intubation of the trachea, preferably with the bronchoscope, in the case of beginning obstruction converts an emergency into an orderly procedure. If one is routinely prepared to do this, he will be repaid for his efforts by lessened operative difficulties. The main point is the use of the Mosher punch tracheotomy in cases which have to be left in more or less inexperienced hands. I do not feel that this procedure has ever had the recognition that it deserves. Its use will oftentimes give a greater sense of security to the operator and less anxiety to the attending physician.

PROFESSIONAL BLDG.

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Clinical Notes

LXV

CARCINOMA OF THE ESOPHAGUS: ENDOSCOPIC EXPERIENCES DURING THE PAST YEAR

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Widespread knowledge of the possibilities of esophagoscopy has resulted in the medical profession's seeking this aid in all cases in which the diagnosis is in doubt. Therefore, we are being asked to examine more cases than formerly, and frequently soon after the onset of symptoms. It is highly desirable that carcinoma of the esophagus be found sufficiently early so that surgery may be feasible. One of the conditions encountered is the border of firm, fixed edematous tissue that lies just proximal to the actual lesion. This edematous tissue is covered by normal-appearing epithelium and may be of such size that to bite through it would be dangerous. The esophagus is usually narrowed by this tissue so that it is practically impossible to see or get at the tumor itself. A small esophagoscope can be inserted in the large one and through the strictured area, or inserted alone. However, this narrows the field of vision greatly and necessitates a much smaller biopsy forceps. Also the narrow area with its rigidity limits the mobility of the esophagoscope considerably. Hence one is often faced with the choice of either making a diagnosis from the local findings or taking a chance of perforating by biting deeply into the tissue for biopsy. Taking the usual bite into the edematous tissue leads to a negative diagnosis for carcinoma. The endoscopist carries considerable responsibility when it is realized that an early positive diagnosis may make surgery possible, whereas an indeterminate report may cause the thoracic surgeon to fear exploration and as a result of waiting the case may finally become too far advanced for surgery. Another situation encountered in taking a biopsy is the carcinoma with a proximal firm border of epithelial hyperplasia. Because of this it is sometimes advisable to take several biopsies and if possible from different sites. There are times when re-examination

of the esophagus is not possible and the exact diagnosis of the case rests on the single esophagoscopy and biopsy taken at this time.

One of the difficulties of esophagoscopy is the frequent lack of time for adequate preparation of the case. Patients are referred for endoscopy, and it is asked that the procedure be carried out that day or the next. The history is briefly given and the patient seen just before the scheduled examination. It would be much better, whenever possible, for the endoscopist to have a chance to take his own history, see the barium fluoroscopy and study the pictures. Some of this may even bear repeating. Then with these things in mind an intelligent esophageal examination may be carried out. Furthermore, in the use of local anesthesia for esophagoscopy it is advantageous to know the size, weight and temperament of the patient.

The following eight cases are reported briefly:

REPORT OF CASES

CASE 1.—Frank C., aged 57, complained of loss of weight for two months, anorexia, and a shaking sensation of the body on swallowing food. He had been told years previously that there was something wrong with the position of his stomach, and he had had discomfort in the chest for many years. X-rays revealed a congenitally short esophagus with the stomach lying in the right thorax. There was also some dilatation of the esophagus just above the cardia, at which site there was pathologic narrowing. On esophagoscopy an irregular grayish, ulcerating, bleeding lesion was found 33 cm. from the incisor teeth. Biopsy revealed squamous epithelium, with apparently epidermoid carcinoma. Surgery was proposed but the patient refused. Death occurred seven months later. Autopsy revealed the short esophagus (13 cm.) and the stomach lying in the right chest. Study showed a large adenocarcinoma of the stomach extending to the cardiac orifice. The case is to be reported later from the gastric standpoint.

CASE 2.—James O., aged 65, had difficulty in swallowing for one month. There was no pain, although food seemed to lodge in the throat. On X-ray examination thick barium lodged in the lower third of the esophagus but passed onward before a satisfactory picture could be taken. The first two esophagoscopies showed indefinite findings, but the third and fourth revealed a narrowing at 38 cm. from the incisor teeth. The mucosa bled easily, and the tissue was firm and fixed. Examination of the biopsy specimen showed fibrinous exudate and evidence of chronic cellular reaction. The patient's general condition was poor and nothing other than a special diet was prescribed. Death occurred eight months later at the Cook County Hospital. The hospital report states the case was diagnosed as a carcinoma of the esophagus, but no postmortem was done.

CASE 3.—Frank M., aged 35, was referred by Dr. Bernauer. The patient stated that he had begun having difficulty in swallowing food two months previously. Food appeared to block at the mid-sternal level. At present only water, milk and eggs could be swallowed without regurgitation. The patient had lost 50

pounds in weight. There was a history of diabetes and alcoholism. Fluoroscopic examination with barium showed a narrowing at the level of the seventh and eighth dorsal vertebrae. Because of the smooth borders the cause was in doubt. Esophagoscopy revealed the narrowing at 30 cm. from the teeth, with some dilatation above and smooth, pale, pasty mucous membrane. The narrowed region was firm and rigid but with a smooth, pale mucosa. The orifice was to the left and a No. 13 French bougie passed through it. The biopsy specimen, although showing inflammatory reaction, showed no carcinoma. The patient dropped from sight, turning up several weeks later at the Billings Hospital. Here Dr. Lindsay was able to get sufficient evidence on a second biopsy to diagnose carcinoma. Operation was carried out, the tumor being removed. It was found attached to the aorta and the trachea. The day after the operation the patient died due to postoperative rupture of the trachea. Postmortem examination showed no evidence of metastases.

CASE 4.—Stanley O., aged 48, complained of dysphagia for three months. Food was regurgitated, there was a discomfort in the neck and chest, and a loss of weight of 20 pounds. X-ray showed a filling defect at the middle third of the esophagus. On esophagoscopy there was a firm, bleeding mass at 32 cm. from the teeth. A No. 16 French bougie could be passed through the narrowing. Biopsy revealed epidermoid carcinoma. The case was discussed at the tumor clinic, but because of apparent extravasation of barium seen on the X-ray film surgery was not advised. The patient returned home, and gradually lost ground until death supervened, five months following esophagoscopy. No autopsy was performed so far as is known.

CASE 5.—William B., aged 57, was referred by Dr. H. Sheaff. The patient had had intermittent hoarseness for two months. Examination showed periodic paralysis of the left vocal cord. X-ray of the chest was inconclusive, and while barium injection revealed partial obstruction of the esophagus the exact diagnosis was impossible. Esophagoscopy revealed a definite narrowing about 26 cm. from the incisor teeth. The lesion was firm and rigid, but covered by normal mucosa. No biopsy was taken. A second and third esophagoscopy were carried out in an attempt to reach the actual underlying pathology, but with little success. Several biopsy specimens were taken but proved negative for malignancy. The patient therefore received deep X-ray therapy for several weeks. Death occurred in a short time (two months following esophagoscopy), and autopsy showed a large carcinoma of the esophagus at the level of the trachea. It had infiltrated and penetrated this structure, thus causing a terminal aspiration pneumonia.

CASE 6.—Charles W., aged 66, noted difficulty in swallowing for six months. There was no pain or regurgitation, but the condition became progressively worse and food had to be swallowed slowly. The roentgenogram showed a napkin-ring filling defect of the esophagus just beneath the upper border of the sternum, probably due to a neoplasm. The esophagoscope was arrested 25 cm. from the incisor teeth by a firm, red, irregular, bleeding obstruction. It was impossible to proceed further as the passage was narrowed to a mere slit. Biopsy revealed a papillomatous epithelium but no invasion below the basement membrane. The patient had a gastrostomy at the County Hospital because of obstruction and died there a few days later. No autopsy was obtained.

CASE 7.—Mike S., aged 49, was referred by Dr. Graf. The patient complained of regurgitation for three months, beginning one day after eating sausage meat.

Lately there had been pain in the diaphragm region. X-rays showed a filling defect just above the diaphragm, probably a malignancy. On esophagoscopy a firm, red, bleeding lesion was encountered at 45 cm. from the incisor teeth. Biopsy revealed epidermoid carcinoma, Grade III. Exploratory operation was carried out by Dr. Dorsey, but the presence of metastases to the liver precluded resection of the esophageal lesion. The patient steadily lost ground, dying several months later. No autopsy was obtained, but at the time of death large masses could be felt in the upper abdomen.

CASE 8.—August D., aged 57, noted difficult swallowing for two months, and was able to get down only liquids and soft foods. On X-ray there was a napkin-ring filling defect in the middle third of the esophagus, probably a carcinoma. Esophagoscopy revealed a firm, red lesion on the anterior wall at 30 cm. from the teeth. A No. 16 French bougie was passed beyond the narrowed portion. Biopsy showed a Grade III undifferentiated carcinoma. The case was considered unsuitable for operation at the tumor clinic because of apparent extravasation of barium beyond the confines of the esophagus. The patient died a month later at the County Hospital from a terminal lung abscess. No postmortem was obtained.

DISCUSSION

A study of these cases brings out various points of interest. All eight patients were men, showing the preponderance of males.

The average age was 54, the youngest, 35, the oldest, 66. Occupation apparently played no part in the condition. The man aged 35 had a definite history of alcoholism. The majority of patients noticed symptoms one to three months before coming in for medical examination. Only one man had noted symptoms for six months, while another noted hoarseness first and the dysphagia for only two weeks. The Wassermann was negative on the five patients on whom it was taken. X-ray examination pointed strongly to carcinoma in four cases, while in the other four, changes were noted but further information was needed to make a diagnosis. The importance of endoscopic examination is thus indicated. Esophagoscopy confirmed the X-ray as to the site of the lesion, three being at the lower end of the esophagus, the others being approximately in the middle third. The aorta, and the trachea or left main bronchus frequently become involved in this latter group. All of the cases were clinically malignant on esophagoscopy, and four were confirmed by a positive biopsy (epidermoid carcinoma ungraded in two cases, and in two cases, Grade III). Of the remaining four, two were proved positive on autopsy later, while the last two followed the typical course of a malignancy.

As to treatment two patients were operated on. One revealed metastases to the liver so nothing further was done, while in the

other the growth was resected but the tracheal wall gave way in 24 hours. One patient had a gastrostomy because of the obstruction. Palliative X-ray treatments were given to only one patient and without improvement. The remaining four patients were put on special diets of high caloric value. However, the end result in all eight cases was fatal. Five died with obstruction, anorexia and increasing weakness, two patients developed fatal pulmonary complications, and there was one postoperative death. The longest interval of time between the onset of symptoms and death was one year, while the shortest was eleven weeks. This last patient was the one who noted hoarseness first and dysphagia for only two weeks prior to examination. The case illustrates how well-developed carcinoma of the esophagus may be present with practically no symptoms of this organ until too late for surgery. The length of time from esophagoscopy until death varied from five weeks to seven months. In none of the eight cases did esophagoscopy or biopsy cause any harm or complications.

In general the points brought out by this study serve to emphasize experiences in these cases. Carcinoma of the esophagus should be borne in mind in all doubtful cases and should be carefully sought for by all possible methods. It is advantageous wherever possible that these patients be cared for by a group of men who are accustomed to working together. Such groups will be more likely to arrive at an early accurate diagnosis and an effective treatment than individuals working occasionally together.

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LXVI

THE EFFECT OF THIAMIN CHLORIDE ON CARDIOSPASM AND ACHALASIA OF THE ESOPHAGUS

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January 8, 1938, a boy was admitted to the Baptist Memorial Hospital with a septicemia and mastoiditis. He later developed pneumonia with empyema, and a paralytic ileus. For several days the abdomen, was greatly distended, very tender, typmpanitic, and extremely painful. There was no bowel movement, and the food intake was practically nil. Recalling the effect of Vitamin B-1 deficiency on the motility of the intestines, he was given 2,000 units of thiamin chloride intramuscularly. That evening he had an excellent bowel movement with some relief of distention. There being no further bowel action for 48 hours, the B-1 was repeated with the same result and the distention rapidly disappeared.

In reviewing the literature on the effect of avitaminosis B-1 on the motility of the bowel, I was struck with the marked analogy between that and the findings in cases of cardiospasm which have come to autopsy. The comparison is given in the following table:

Avitaminosis B-1	Cardiospasm
Anorexia and dilatation of the colon	Dilatation of the esophagus
Degeneration of Auerbach's plexus	Degeneration of Auerbach's plexus
Ptosis of the diaphragm	Ptosis of the diaphragm
Visceroptosis	Visceroptosis
Gastric atony and intestinal stasis	Esophageal atony and stasis
Atrophy of muscles with fibrous contractions.	Atrophy of muscles with fibrous contractions.

I was fortunate in acquiring two cases of cardiospasm in the next few months which were subjected to a clinical trial with thiamin chloride.

Case 1.—Mrs. A. W. was referred by Dr. J. C. Ayres, June 23, 1938. During a G. I. examination for symptoms suggesting chronic appendicitis, a marked cardiospasm was discovered. July 1,

1938 the diagnosis was confirmed by esophagoscopy. Dilatation resulted in only partial and temporary relief.

July 8th, 3,000 units of thiamin chloride were administered. This was repeated July 12th and July 15th. There was complete relief; the patient could eat any food. Thiamin chloride was given by mouth and there has been no recurrence of the cardiospasm to date.

CASE 2.—A. C. W. was referred by Dr. Giles Coors, June 25, 1938. The patient complained of difficulty in swallowing fluids. Fluoroscopic examination with barium showed constrictions at the level of the aorta and at the cardia. Esophagoscopy demonstrated cardiospasm—only partial relief was obtained from dilatation. The esophagus was again dilated June 30, 1938, with still only partial relief.

July 4th, 3,000 units of thiamin chloride were administered. This was repeated July 8th, July 11th, and July 14th. There was complete relief. Thiamin chloride was given by mouth, with no recurrence to date.

It might reasonably be argued that improvement in these two cases was the result of the esophagoscopy rather than the administration of Vitamin B-1. Hence the following two cases:

CASE 3.—W. C. J. was referred by Dr. L. C. Sanders and Dr. J. F. Hamilton. This patient, while confined to the Campbell Clinic with an extremely painful arthritis of the hip, developed difficulty in swallowing. For two days prior to my examination he had not even been able to swallow water and complained of severe pain in the esophagus on attempting to swallow. Having developed an acute pulmonary condition with fluid in the left chest, he was transferred to the Baptist Memorial Hospital where I first saw him. X-ray with barium showed marked cardiospasm. This was not relieved by aspiration of the chest. Because of the hip condition, esophagoscopy was not performed.

6,000 units of B-1 were given intramuscularly at 5:00 P. M. In a few minutes he became comfortable and began swallowing fluids. His diet was gradually increased, and he had no further difficulty in swallowing, up until the time he left the hospital.

It is interesting to note that in this case, at least symptomatic relief was obtained in spite of the definite pathology in the left lung and thorax.

My most spectacular case is as follows:

CASE 4.—Mrs. G. B. was referred by Dr. Max Henning. The patient was first seen in October 1934. While undergoing a gastrointestinal X-ray, a cardiospasm with an enormous dilatation practically filling the thorax was discovered. There was a history of regurgitation for several years. The patient had been forced to sleep on three pillows because food ran back up into her throat. Esophagoscopy with dilatation did not relieve her. She went to Jackson in Philadelphia and had been using a mercury-filled bougie daily since then.

October 17, 1938, she was given 3,000 units of B-1 intravenously after a meal. She stated that in a few minutes she had a sensation of contraction in the esophagus and felt the food slip through into the stomach. For the five days that she was in Memphis she was given 3,000 units once a day, intramuscularly, after the big meal, and during this time she did not regurgitate or require the use of the bougie. She was sent home with instruction to have 3,000 units administered daily for a month and then return for a barium X-ray. A year later the patient reported that she was still swallowing without difficulty although it has been necessary for her to continue the use of thiamin chloride hypodermically.

Mosher, particularly, has been dissatisfied with the extent of our knowledge of the cause of cardiospasm, continuing to investigate even after establishing two separate and apparently logical theories of the cause. In as much as he has probably devoted more attention to cardiospasm than any other investigator, I believe that quoting his views on the subject will give a representative idea of the accepted theories today.[1]

Mosher states that there are two chief causes of cardiospasm: 1.) Stricture of the terminal portion of the esophagus, usually a single one, somewhere in the cone of the diaphragm, through the whole liver tunnel and the cone of the diaphragm which it encloses and supports, may be narrowed. The commonest site of the stricture is at the twist between the vertical and horizontal arms of the terminal portion of the esophagus. Many of the strictures at this point are but a slight gluing together of the folds of the mucous membrane. A very slight inflammatory process will produce a gluing of the folds of the esophagus at the point of the twist, as the twist is a predisposing factor. 2.) Mobilization and displacement of the terminal portion of the esophagus through ptosis of the diaphragm, which releases the terminal portion of the esophagus from

the support of the cone of the diaphragm, resulting in the formation of a trap at the lower end of the esophagus. Mosher states that most cases of cardiospasm have a fallen or ptotic diaphragm, along with a general viceroptosis. The falling of the diaphragm opens up the cone of the diaphragm and sets the terminal portion of the esophagus free to sag and twist as it may.

Later, after accumulating more autopsy material, he modified his views, to the belief that as a result of infection and inflammation, either from the thorax or the abdomen, in the connective tissue filling the diaphragmatic cone, bands and adhesions are produced which constrict the terminal portion of the esophagus or distort it by traction. With the destruction of the motor cells (Auerbach's plexus) by infection, the muscles of the esophagus lose their power to contract, peristalsis is progressively lost and the walls of the esophagus after a period of preliminary hypertrophy become thin, and the whole of the esophagus with the exception of the terminal portion gradually dilates.

Mosher also states, that he has a growing conviction, that the obstruction in the terminal portion of the esophagus, in cases of cardiospasm, is a slight affair.

Unquestionably, Mosher has ably and completely described both the pathologic findings in cardiospasm and the mechanics by which it is produced. It was my belief, however, that in many cases the initial cause of cardiospasm may be avitaminosis B-1 rather than infection, and on the basis of the clinical experiences in the case reports detailed above, I developed the following theory as to the steps in the development of cardiospasm.[2]

Anorexia, the first symptom of B-1 deficiency, leads to a greater deficiency. An early finding in B-1 deficiency being dilatation of the colon, we can assume the same occurs in the esophagus. With dilatation of the esophagus and loss of motor stimulus through degeneration of Auerbach's plexus, stasis of food follows as the result of loss of the propulsive force of the esophageal walls rather than by reason of constriction at the cardia or in the liver tunnel. Adhesions between the folds of the mucosa develop through maceration of the mucosa caused by detritus lying in the esophagus. Ptosis of the diaphragm, developing by reason of the B-1 deficiency, further complicates the picture, so that in the advanced stages we find all the factors described by Mosher increasing the obstruction but occurring as secondary factors. A logical explanation of why cardiospasm is benefited by dilatation if it is primarily due to loss of propulsive

force, is that dilatation enlarges the outlet so that food is forced through by a much weaker contraction of the esophagus than normally; in turn the patient returns to a normal diet, thus correcting the vitamin deficiency and providing the necessary stimulus to the propulsive force in the form of Vitamin B-1.

On this basis, the following conclusions may be drawn:

1. The primary obstructive factor in cardiospasm is the lack of propulsive force in the falls of the esophagus rather than obstruction at the cardia.

2. Mosher's intrinsic findings can be the result of stasis in the esophagus and his extrinsic findings, the result of Vitamin B-1 deficiency, rather than the primary cause of cardiospasm.

3. Cardiospasm is frequently a manifestation of avitaminosis B-1.

4. Vitamin B-1 given intramuscularly will relieve an attack of cardiospasm in many instances.

5. Continued administration of Vitamin B-1 will prevent recurrence of attacks of cardiospasm in these cases.

After publishing my preliminary report,[2] my attention was directed by Dr. Mosher to the work of Eduardo Etzel of the University of Sao Paulo, Brazil.[3] To Etzel unquestionably is due not only priority in establishing avitaminosis B-1 as a cause of cardiospasm, but also the greatest praise for the completeness of his neuropathologic studies. His only publication in the English language appeared in Guy's Hospital Reports in 1937, but he has published several additional reports in Brazil. His conclusions and findings are abstracted freely because they should be incorporated in our literature.

Mega-esophagus is caused by chronic lack of Vitamin B-1. It is a symptom of a degenerative disease involving the entire autonomic intramural nervous system, which, once established, can be relieved but not permanently cured, as the autonomic nervous system does not regenerate. Associated with mega-esophagus are frequently found megacolon, anachlorhydria and hypochlorhydria; cardiac and electrocardiographic changes; infrequently polyneuritis; dysmenorrhea; a lowered basal metabolism; and achalasia of the ureter with hydronephrosis and hypertrophy of the bladder.

The neuropathology is demonstrated to be a degeneration of Auerbach's plexus, distributed throughout the entire length of the

esophagus and not confined to the lower third, as would be expected if it were the result of esophagitis.

To anyone interested in cardiospasm, reading Etzel's articles in full will well compensate him for the time spent.

Avitaminosis B-1 has been definitely established as a cause of cardiospasm, and there are many cases, particularly if seen in the early stages, that will respond to treatment with thiamin chloride. In conclusion, a note of warning must be sounded. Every obstructive lesion of the esophagus must be subjected to an esophagoscopy. Response to thiamin chloride is not sufficient evidence upon which to base a diagnosis. I have seen one case in whom the initial obstruction was relieved for three months by the administration of thiamin chloride, but an esophagoscopy at the end of this period revealed carcinoma.

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LXVII

(From a Group of Case Records Collected and Abstracted Under the Title of "My Most Interesting Case," By Noah D. Fabricant, M.D., Chicago)

TRIFACIAL NEURALGIA

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My most interesting case in the specialty of otolaryngology dates back to the days when I dabbled in borderline surgery about the ear, nose and throat—and the resection of a Gasserian ganglion was a more than welcome morsel on an operating day. This patient of mine had a true tic that failed to respond to any type of treatment. I finally decided to do a ganglionectomy, at the time a preferred operation, as recommended by Krause-Cushing. During the exploration of this structure I ruptured the middle meningeal artery, or perhaps a larger vessel, and all I could do in the face of this was to make use of gauze packing. Certainly I used about a yard of gauze, six inches wide and about one-quarter of an inch thick, but the bleeding still continued. I then hurriedly exposed the common carotid artery and ligated it. Upon removing the gauze packing bleeding ensued, so the gauze packing was resumed and the wound closed. The patient had no symptoms of paralysis and very little evidence of intracranial pressure. He obtained considerable relief from pain, although the ganglion had not been touched during the operation.

My conclusion was that I had probably ruptured either the internal carotid artery or an aneurysm. A complete study of the spinal puncture and fluid analysis interested me greatly. In the first place, both the fluid and the pressure before operation had been absolutely normal. One day following operation, and with that massive gauze pack still present intracranially, the spinal fluid was greatly under pressure and contained a large amount of blood. Remembering that there was no opening in the dura, I could explain the presence of blood in the spinal fluid only on the basis of extreme passive congestion or perhaps on the basis of a small rent made by the packing. This marked admixture of blood made most of the other cerebral spinal fluid tests useless. However, what was most interesting was the find-

ing that before operation the Lange gold colloidal test was negative while after operation it was positive. At no time during several repeated fundus examinations was there any evidence of swollen discs.

In looking for an explanation I corresponded with Cushing of Boston, Frazier of Philadelphia and others. Their views varied so much that I now hesitate to put them into print. Suffice it to say that all of these men agreed that the disappearance of the tic and the pain following my intervention was undoubtedly due to some destruction produced by the pressure of that massive gauze pack. However, some time later the patient died, and since a postmortem was not permitted, the mystery concerning my most interesting case was never completely solved.

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THE STORY OF WARREN BELL

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In this day of ultra-modern surgery multiple operations create little but passing interest. Many patients boast of having 10 or 15 operations, few have had more than 20 or 30, and rarely does one encounter a patient who has had 50 or 75 operations. Warren Bell exceeded the above figures and totalled the incredible number of 97 operations—each under a general or intravenous anesthetic. How one frail body could withstand the repeated punishments inflicted by repeated anesthetics and operations has been difficult to understand, yet the need and hunger for oxygen and the determination to live spurred the courage of this youngster to submit to these numerous procedures.

On July 17, 1917, when Warren Bell was only two and one-half years old, he was referred to a laryngologist because of increasing difficulty in breathing. An examination revealed the presence of multiple papilloma involving both vocal cords and ventricular bands. There was a subglottic extension of the papilloma. Under a general anesthetic and by means of suspension laryngoscopy the first of many operations was performed. The papilloma were pinched off

with the aid of a Mosher forceps and care exercised not to create any new raw surfaces. For a period of three months there was no difficulty in breathing, but on November 30, 1917, it again became necessary to remove the multiple papilloma which had recurred, and in appearance were similar to those seen at the time of the first operation. At intervals of a few months, operation followed operation until in July of 1918 it became necessary to perform a tracheotomy. However the further extension of papilloma into the trachea continued in spite of every known means to prevent it. Frequent operations at close intervals now were necessary to keep the airway clear. Every known method for the control and eradication of papilloma was used unsuccessfully, and fulguration, scalping cauterization and local applications were employed in vain. Aspiration pneumonia was a dangerous complication in September 1919 but was successfully conquered. The fight against papilloma continued, and by July 1920, 32 operations had been performed. After repeated consultations it was deemed advisable to employ radium so as to help control these stubborn growths. This was done by using 50 mg. radium, moving it up and down over the papillomatous masses for a period of 30 minutes. Four months later the growths appeared less abundant, and radium was thought to be responsible for the apparent decrease.

At this time fulguration was used and radium again applied. One month later examination disclosed the happy fact that there was little or no growth below the cords. Again radium was used. In May 1921 the enthusiasm over the accomplishments of radium received a tragic blow. The development of a tracheoesophageal fistula as a direct result of a radium burn necessitated a gastrostomy. Meanwhile papilloma again developed beyond the tip of the tracheotomy tube and gradually extended to the bifurcation of the trachea. It was imperative that these be frequently removed to keep a clear airway. Numerous plastic closures were attempted on the tracheoesophageal fistula, with ultimate success in 1925. Radium necrosis slowly destroyed the greater part of the thyroid and cricoid cartilages, leaving the larynx open externally with a gutter-like appearance. In this hollow, papilloma grew very rapidly and extended into the hypopharynx. A second complication of the radium burn was the development of an esophageal stricture which required frequent dilatations. Warren Bell was now approaching the period of adolescence, a time we welcomed since eminent authorities stated emphatically that multiple papilloma of the larynx were self-limiting and always disappeared at this age. Warren, however, was destined to be the exception to the rule—though it must be stated that the

operations were performed at much longer intervals, a fact that showed that the growth of papilloma had materially slowed down.

The constant hope that each operative procedure would be the last and the obstinate papilloma completely eradicated continued within us. While hope sprang eternal, papilloma continued eternally to grow from the trachea, extending not only to the bifurcation but involving the rim or orifice of the right bronchus. A large mass of papilloma was always present at the tracheal opening and the gutter-like cavity which represented the location of the larynx. At times this mass became as large as a full-sized lemon. An average of only three operations per year from 1926 to 1936 was necessary to keep Warren comfortable. During this time daily esophageal dilatations with No. 16 and No. 18 French bougies were done. In December 1935 papilloma protruded into the lumen of the esophagus at the site of the former tracheo-esophageal fistula. In November 1936 many less papilloma appeared in the trachea, but the growth seemed more abundant in the region of the larynx, protruding externally as well as partially filling the hypopharynx, and produced some dysphagia. Removal of the entire mass was considered, a procedure which would leave a fistulous opening into the hypopharynx with a diameter of about two inches. It was deemed unwise to establish such an opening at this time. Because of the gross pathological changes observed in the mass a biopsy was submitted for histological study. Dr. Lanford, pathologist at Touro Infirmary, reported "some areas show definite infiltration into deeper portions of the stroma. However, the component cells are well differentiated and at the present time are doubtful of metastasizing properties. Histology warrants the diagnosis of papillary carcinoma, Grade I."

Warren Bell was now 21 years of age. He was tall, thin and rather pale. Between operations he had attended school as often as he could. In spite of the loss of his larynx Warren had developed and excellent voice, somewhat whispered in character, but with good tone and loud enough to be heard easily all over the classroom. When his breathing permitted he engaged in baseball and basketball; in spite of his handicap he was socially very attractive. A devoted mother supplied his every want and need. Mother-love manifested itself in its truest form. She was a devoted nurse to the very end.

Early in December 1936 progressive loss of weight and inability to swallow, due partially to occasional dilatation of the strictured esophagus, necessitated a second gastrostomy by Dr. Urban Maes. At this time an examination of the trachea disclosed very few papil-

loma. There was in the region of the hypopharynx, however, an area of infiltration involving the mouth of the esophagus; externally this mass completely filled the former site of the larynx infiltrating the surrounding tissue. An attempt at removing part of the growth was made in order to create a fistulous opening to the hypopharynx. From the gross appearance it was quite evident that the original growth had undergone malignant degeneration. This was confirmed by the pathological report of squamous cell carcinoma Grade II. Following this, his 97th operation, Warren developed an aspiration pneumonia and quietly expired on November 19, 1936.

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PHARYNGEAL HEMORRHAGE

Samuel J. Pearlman, M.D.

Chicago

About ten years ago I had a patient under my care who presented a series of findings that were new to me and to most of the men in our specialty with whom I had discussed the case. My patient was a male child, about two and a half years of age, who started out with an acute tonsillitis and upper respiration infection and within a week developed a rather marked swelling involving both sides of the neck, particularly the right side. Breathing was difficult, noisy and through the mouth. Examination of the pharynx revealed a pushing-in of the lateral wall of the pharynx and soft palate on the right side. The neck swelling was taken to be a severe periadenitis and cervical cellulitis in which no fluctuation was demonstrable. Because of the stormy course and the septic temperature, the child was hospitalized with the hope that pus would be present and that incision and drainage would change the course of events.

After two or three days of observation, however, there was still not much improvement. Hoping to find pus even though there was no softening, all of the tissues of the pharynx and in the neck remaining stony-hard, a forceps was thrust into the right-sided lateral pharyngeal swelling, but no pus was encountered. The next two days saw little change in the patient's condition. Our anxiety was not diminished, but inasmuch as this was before the days of widespread use of roentgenograms for conditions of this character and long

before the use of sulfanilamide, there was little that we could do beyond the application of heat. Forty-eight hours later I was informed by the interne—I had seen the child a few hours before—that the child suddenly sat up, a severe hemorrhage from the pharynx ensued, and death came on within several minutes. Since permission for postmortem examination could not be obtained the cause of the hemorrhage remained undetermined.

Some time later I saw a case similar to the one just described. This concerned a five year old female child who, following a sore throat, had developed fever and swelling of the neck for the past seven days. The swelling of the neck was diffuse, painful on the left side, and spread up to the face at the angle of the jaw. No single nodes were palpable. There existed a fullness on the lateral wall of the pharynx on the left side.

The child was hospitalized. With no previous manipulation such as incision, the child awoke suddenly from sleep and spit up a large amount of bright red blood. The patient went into shock, and the pulse became imperceptible. Although the bleeding soon stopped, the respirations remained rapid and shallow, and the patient complained of considerable thirst. External heat, ice collar to the neck and other usual remedies were applied. In view of the amount of blood lost, the patient's condition and the knowledge of the story of the first case, the attending physicians were in all likelihood impelled to order a transfusion of blood and to consider imperative the ligation of the carotid artery.

Because of the swelling at the level of the bifurcation of the carotid and the patient's condition, our inability to reach the external carotid forced us to ligate the common carotid, inasmuch as it was the only one accessible. The child made a more or less uneventful recovery, and it was this experience that aroused our interest in investigating the literature. Here was discovered the answer to many of the problems that were not clear. To our surprise we discovered that our original notion that a lesion of the external carotid was responsible for these severe hemorrhages was wrong. Previously it had seemed natural to think that in an infection springing from the tonsils and involving the adjacent neck tissues some branch of the external carotid was the offender. From the literature it became clear that in many cases of severe hemorrhage springing from the pharynx and incident to peritonsillar abscess complicated by involvement of the parapharyngeal space, retropharyngeal abscess complicated by involvement of the parapharyngeal space, and primary parapharyngeal space infections, the internal carotid artery and the common

carotid artery were eroded mainly. This was due to the fact that the primary lesion was a severe adenitis and that these inflamed glands were so situated as to attack these vessels with the greatest of ease.

Hemorrhages following only in the course of complicated peritonsillar abscesses were apt to come from branches of the external carotid artery, and it is only in this type of case that seeking and ligating this vessel is justified. It then became plain that the thing we had not wanted to do, namely to imperil the brain by ligating the common carotid artery, was precisely the measure that it was essential to do; and that hemorrhages of this kind so severely threaten a patient's life that the risk of brain complications must be assumed. Further, we discovered that the risks of common carotid artery ligation under these circumstances were not nearly so great as was generally believed. For this procedure, done as it is under circumstances we have described and as a rule in young people and children, carries with it a relatively small morbidity and mortality. When one considers that hemorrhage following retropharyngeal abscess—and this occurs most often in children under two years of age—has a mortality of 100% in those cases where ligations of the carotid are not carried out, and when we are able to save some patients when ligation is undertaken and see the improvement that follows surgical intervention, there can be no doubt of the advantage obtained when ligation is performed.

55 E. Washington St.

A CASE OF CARCINOMATOSIS OF THE MENINGES

Hans Brunner, M.D.

Chicago

A 58 year old male had been entirely well until September 22, 1939, at which time he suffered a dizzy spell followed shortly by a feeling of malaise. That night he had numerous dizzy spells, complained that the room rotated around him, and vomited. The next day he continued to have more dizzy spells and periods of transient vomiting.

Three days later he noticed a ringing sensation in both ears. This was rapidly followed by progressive deafness so that within a

week he was completely deaf. He also observed that when he walked he inclined to either one side or the other, more often to the left. During these dizzy spells he experienced a sensation of falling, but he was unable to describe the direction of the falling. These dizzy spells grew progressively worse until the patient had a number of attacks daily, not always associated with vomiting.

His visual acuity failed rapidly during the next three weeks, and the printed page became blurred. When he attempted to walk he felt unsteady and frequently grasped at some object for support. Occipital headaches and dryness of the mouth developed.

The neurological examination on October 16, 1939 revealed the following: slight haziness of the right disc; increased deep reflexes on the left side; decreased abdominal reflexes on the left side; nystagmus on lateral gaze; total bilateral nerve deafness; and a right lower facial palsy. The spinal fluid, blood chemistry and urinalysis were negative. A roentgenogram of the skull showed a suspicious lesion at the top of the cranial vault. An encephalogram was done, but no pathology was revealed.

The otological findings were as follows: drums slightly retracted; deafness of both ears; labyrinth not irritable, neither for caloric nor for tuning test. There was slight stiffness of the neck.

Because of the history, the ear findings and the stiffness of the neck I made a tentative diagnosis of carcinomatosis of the meninges. I suggested that a thorough investigation be made for a primary lesion either in the lungs, lower digestive tract, or prostate. Further examination revealed a carcinoma of the rectum, substantiated by biopsy. At first it was believed that the carcinoma at this site should be resected, but since the patient was rapidly going down-hill the surgical consultant advised against resection at this time. Accordingly, in view of the roentgenogram findings and the rectal findings, a positive diagnosis of carcinomatosis of the meninges was now made. The patient continued to do poorly, and it became necessary to feed him by nasal tube. On November 17, 1939 he expired. Necropsy verified the clinical diagnosis.

307 No. Michigan Blvd.

FIBROMYOMA OF THE LARYNX IN AN 82 YEAR OLD MALE

M. F. Arbuckle, M.D.

St. Louis

I am reporting a case which is important and unusual from many standpoints. In the first place, the patient on admission was 82 years of age. He had had respiratory and phonatory difficulty over a period of six months. His physical examination was negative except for the findings usual at that age.

With a mirror one could see a large shiny mass that resembled a cyst, filling the hypopharynx and involving the epiglottis. Because of his age and the general weakness which accompanies such advanced age he was sent to the hospital with the idea of incising the cyst and draining its contents. When a knife was inserted through the laryngoscope the cyst proved to be a solid tumor without liquid content; its interior was everted through the incision. This was done by the aid of local anesthesia. There was some interference with respiration. The bronchoscope was inserted immediately for respiratory relief, and the patient etherized through it. Then an endotracheal tube was placed in position, and the patient was suspended with the Lynch apparatus. A tumor accordingly, was removed from the external surface of the larynx by blunt dissection. Its attachment extended along the rim of the glottis on the right side back to the midline and downward to the level of the cricoid cartilage. The pharyngeal surface of the larynx on the right of the midline was denuded by the necessary dissection from the rim of the glottis to the level of the cricoid. The tumor was the size of a hen's egg and came away in three pieces. There was surprisingly little bleeding and that which did occur was easily controlled. The patient was returned to bed in good condition.

Twenty-four hours later edema of the larynx necessitated tracheotomy. This was done with a bronchoscope in position and by means of local anesthesia. After the tracheotomy a feeding-tube was inserted because of difficulty with deglutition.

The patient then developed retention of urine and required catheterization. Dr. D. K. Rose's examination revealed a tremendously hypertrophied prostate. After a sufficient period of postoperative recovery from the laryngeal operation and the tracheot-

omy, Dr. Rose did a transurethral resection of the prostate. For a period of three weeks the patient got along with a feeding-tube tracheal tube and a tube in his bladder.

The microscopic report of the laryngeal tumor was fibromyoma. This is the only tumor of this type involving the larynx that I have ever seen.

The patient was finally discharged from the hospital five or six weeks later. He walked out with all tubes removed and with a practically normal voice. I have heard from him since then, and he is in excellent condition. He has gained some 40 pounds in weight.

539 No. Grand Blvd.

The Scientific Papers of the American Broncho-Esophagological Association

LXVIII

REPEATED SEVERE HEMOPTYSIS NECESSITATING PNEUMONECTOMY*

CARLOS E. PITKIN, M.D.

CLEVELAND

The reasons for reporting this case are the unusual severity of the hemoptyses and the difficulty in determining the cause and source of the bleeding.

The patient, an obese Italian, aged 31, was admitted to Lakeside Hospital on May 5, 1940. His previous health had been good. In January 1940 he had had a sore throat and a non-productive cough. A month before admission the cough was productive of small amounts of bright blood.

Examination showed dullness at the apex of the left lung, posteriorly. Sibilant rales were heard in the left interscapular region and at the left base. The sputum was negative for tubercle bacilli. The blood Wassermann test was negative. Bleeding and clotting time were within normal limits. There was no hypertension. Bronchoscopy four days after admission did not reveal bleeding or any abnormal condition in the trachea or bronchi.

Roentgenograms did not show evidence of pulmonary disease, nor, after lipiodol injection, any bronchial obstruction or bronchiectasis. The day following lipiodol injection there was a hemoptysis of 1250 cc. Bronchoscopy two days later revealed no more than the previous one. There was no further bleeding, and the patient was discharged on June 25.

He was readmitted on July 12, after a small hemoptysis. Bronchoscopy did not show any pathological condition, nor roentgenograms, any evidence of infiltration in the lung fields.

*Presented at the 24th annual meeting of the American Broncho-Esophagological Association, Cleveland, Ohio, June 3, 1941.

He was again admitted on November 25 for observation. His activity had been restricted, and he had had an occasional small hemoptysis. On the day of admission he had coughed up several mouthfuls of bright blood.

Roentgenograms did not show evidence of pulmonary disease. Bronchoscopy on November 30 was negative. He was discharged December 5.

The last hospital admission was on January 20, 1941. Since his previous admission he had not worked. There had been no bleeding until three days before admission, when he had a feeling of a lump in his throat and coughed up a clot and over a quart of bright blood. Since then he had been very weak, had had several small hemoptyses and on the day before admission, one of over a pint. There were three hemoptyses during the next seven days, the first, of 800 cc., the next, of 350 cc. and the last, of 900 cc.

Bronchoscopy three days after the last hemoptysis revealed no active bleeding. A blood clot filled a medial branch of the left lower lobe bronchus. The clot, a bronchial cast 5 cm. long, was removed with forceps. No bleeding was started by suction or sponging.

Pneumothorax was started on February 2. Five days later a hemoptysis of 1100 cc. occurred.

The pneumothorax was maintained, and roentgenograms showed complete collapse of the left lung.

Two weeks after starting the pneumothorax, at 7 P. M. he coughed up 50 cc. of bright blood and at 11:30 P. M. suddenly began to cough up large quantities of blood. This lasted for three or four minutes, when he became very cyanotic and respiration almost ceased. After having the throat cleared of clots he coughed up more clots and was unconscious for 45 minutes.

Bronchoscopy four days after this episode revealed a little blood-stained mucus in the left main bronchus but no bleeding.

During the next ten days he had four hemoptyses, varying in amount from a few to 700 cc.

Bronchoscopy on March 20 revealed a trickle of blood coming from the left main bronchus. It was definitely not from the upper lobe branch, but appeared to come from the branch from which a clot had been removed at a previous bronchoscopy.

Treatment during hospitalization consisted of complete rest, supporting measures and repeated blood transfusions.

After definitely determining that the bleeding was from the left lung, Dr. Samuel Freedlander agreed to accept the patient for pneumonectomy. The left lung was removed by him on April 5.

There has been no further bleeding, and the patient is convalescent in the hospital.

Pathological Report. The lung, weighing 450 gm., shows some thickened rough areas of pleura on the medial surface, also on the anterior edge. The bronchial walls are thinner than normal peripherally and the lumina are broader than normal, but there are no sacular terminal dilatations. The bronchioles are very broad, almost up to the pleura, more marked in the lower lobe. There is no tumor tissue or ulceration. The pulmonary arteries and veins have a normal appearance. At the anterior edge of the left lower lobe there is a wedge-shaped, dark red firm area containing more blood than elsewhere.

Histological Examination. Sections taken from both lobes in many areas show a similar appearance. The alveoli are of normal size and shape. The alveolar walls show no thickening. The most significant change is the presence of large amounts of blood, both recent and old, in many of the alveoli. The distribution of the recent hemorrhage is focal. In some areas the alveoli are empty.

The bronchi throughout present an essentially normal appearance save in the proximal portion where there is a slight fusiform enlargement of the bronchi. The mucosal membranes are intact. The blood vessels throughout are essentially normal. There is no intimal thickening or arteriosclerosis.

Diagnosis: Recent and remote hemorrhage into the alveoli of the left lung. Slight fusiform bronchiectasis. Fibrous pleural adhesions.

I am indebted to Dr. Joseph Wearn and to Dr. Samuel Freedlander for permission to report this case.

CARNEGIE MEDICAL BLDG.

LXIX

THE FUNCTIONAL EXAMINATION OF RESPIRATION IN OBSTRUCTIVE DISEASES*

ALBERT H. ANDREWS, JR., M.D.

CHICAGO

The functional examination of respiration is a procedure which makes possible the recording and measuring of the movement of air in and out of the chest in normal and abnormal conditions. The technique of this examination has been provided previously by Barach and Richards and their co-workers.[1, 2]

The apparatus for this procedure is a modified basal metabolism apparatus of the Benedict Roth type. The essential modification consists of a high speed recording drum thereby demonstrating the qualitative alterations in respiration. The high speed recording can be made by using a two-speed drum or by using two separate drums with an arm extending out from the writing pen holder to support a pen which writes on the second or high speed drum.

A second, although an unessential modification, is an automatic oxygen feeding device[3] which admits oxygen to the apparatus as fast as the patient absorbs the oxygen, thereby producing a horizontal tracing or maintaining a fairly constant oxygen concentration within the apparatus. Due to the fact that the respiratory level of the chest may change during the examination, this addition of oxygen is not always a true indication of the quantity of the oxygen absorbed.

A third modification, the Reichert Ventilograph,[4] records the volume of inspiration only, thus indicating the ventilation or the amount of air inhaled over a period of time. This device, though not essential, simplifies the measurement of ventilation and reduces the time required for calculating the results.

*From the Peroral Endoscopy Clinic and Department of Clinical Research of St. Luke's Hospital and the Department of Physiology and Pharmacology of Northwestern University Medical School, Chicago, Illinois.

Assisted by a grant from the Council on Physical Therapy of the American Medical Association.

Presented at the 24th annual meeting of the American Broncho-Esophagological Association, Cleveland, Ohio, June 3, 1941.

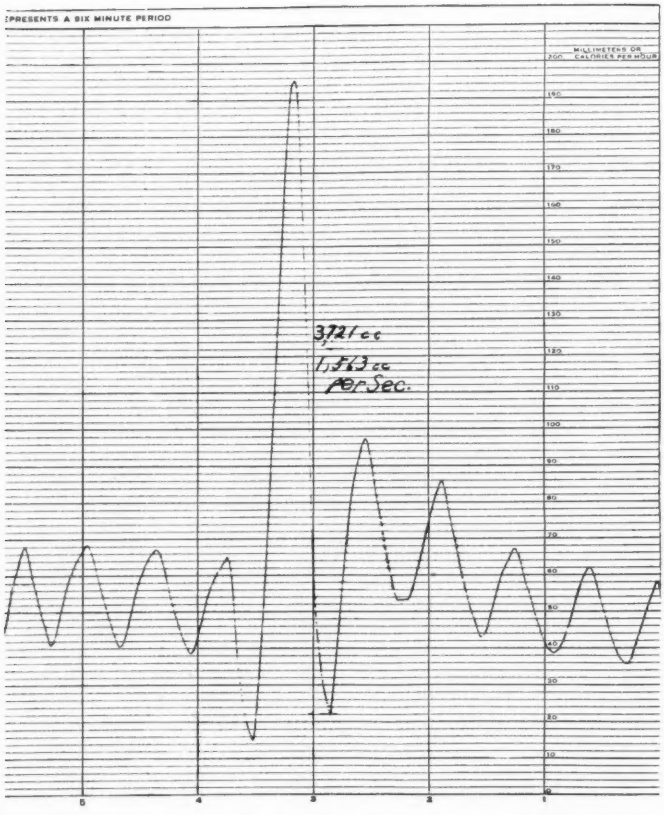

Fig. 1. Normal. (See Table I.) Vital capacity, 3,721 cc., and maximum expiratory air velocity, 1,563 cc. per sec. Note the slight curve on expiration.

The following quantitative characteristics of respiration can be determined by this method of examination: 1) rate of respiration; 2) ventilation or minute volume; 3) rate of oxygen absorption; 4) vital capacity, inspiratory or expiratory and its component parts; complemental, tidal and supplemental airs and the velocity of air flow during the various phases of the vital capacity usually the maximum velocity during expiration; 5) maximum ventilation, i.e., the largest amount of air the patient can breathe in one minute.

TABLE I.—NORMAL SUBJECT

(See Fig. 1)

Rate per min.	12.3
Ventilation cc. Min.	6,935
Oxygen Absorption cc. Min.	280
Efficiency Pct.	4.04
Vital Capacity cc.	3,720
Maximum Expiratory Velocity cc. Sec.	1,560

The qualitative characteristics of the following can be recorded: 1) quiet respiration, 2) vital capacity, 3) maximum ventilation.

The alterations in these factors can be demonstrated as a result of breathing oxygen and helium, of positive pressure and administration of bronchodilator drugs.

RESULTS OF THE EXAMINATION

Normal: Referring to Fig. 1 and Table I, the following characteristics are pointed out for contrast with the abnormal findings: ventilation or minute volume of respiration, vital capacity, maximum rate of expiratory air velocity, general shape of the curves, and absence of air trapping, i.e., failure to expel as much air as is inhaled during successive maximum expirations.

Bronchial Asthma: Referring to Fig. 2 and Tables II and III, quiet respiration shows decreased velocity during expiration and increased ventilation. Vital capacity shows reduced volume and velocity of air flow. The velocity decreased during the latter part of the expiration rather than at the end as occurs in the normal. Air trapping is much less marked than in hypertrophic emphysema or in those cases complicated by emphysema.

Oxygen concentrations of 50% and over usually do not cause much change objectively or subjectively. Helium 80% in oxygen usually shows an increase in ventilation, vital capacity and velocity of air flow without striking subjective improvement. The increase in ventilation indicates that helium is of value inasmuch as the time lag in subjective response is longer than the period of the test. The bronchodilator drugs when effective show objective and subjective improvement.

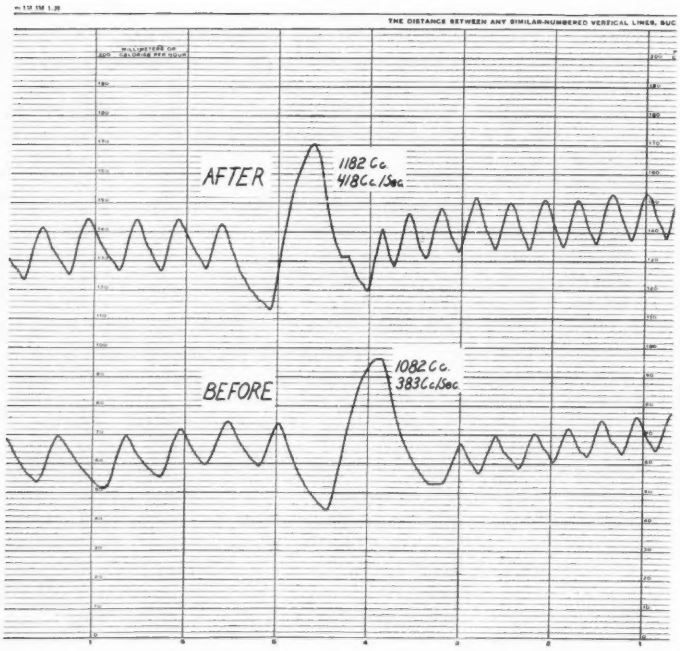

Fig. 2. Bronchial asthma. Shows slowing of expiration, low vital capacity, 1,082 cc., and reduced velocity of air on forced expiration, 383 cc. per sec. Upper tracing shows after inhalation of 1:100 epinephrin: vital capacity increased to 1,182 cc. and increased air velocity of 418 cc. per sec.

Obstructive Hypertrophic Pulmonary Emphysema: Referring to Fig. 3, Tables III and IV, quiet ventilation is increased over the normal with reduced efficiency and the expiratory phase is prolonged. Vital capacity is reduced in volume and maximum velocity of air flow is reduced. The expiratory curve is straighter than in bronchial asthma but more curved than in the normal. Maximum ventilation shows progressive air trapping with the ventilation being accomplished at the inspiratory level. In severe cases maximum ventilation may decrease until it is less than during quiet breathing.

Improvement follows the use of epinephrin and in most cases with the use of oxygen as is evidenced by decreased ventilation during quiet breathing. The response to helium is favorable and similar to the response in asthma.

TABLE II.—BRONCHIAL ASTHMA

(Same Patient as Table III)

	Air	Oxygen	Helium	After Epinephrin Inhalation
Rate	13	14	17	15
Ventilation cc./Min.	8,000	8,820	9,706	11,060
Vital Capacity cc.	1,408	1,709	1,960	2,196
Subjective Relief		+	+	+++

TABLE III.—BRONCHIAL ASTHMA SHOWING RESPONSE TO EPINEPHRIN INHALATION

(Same Patient as Table II)

	11/17/39		12/1/39		2/26/40	
	Before	After	Before	After	Before	After
Rate	13	15	13	15	8	10
Ventilation cc./Min.	8,000	11,060	10,850	12,730	6,615	7,710
Vital Capacity cc.	1,408	2,196	2,550	2,900	3,607	3,518
Maximum Expiratory Velocity L./Min.	369	922	389	891	698	736
Subjective Relief		+++		+		0

TABLE IV.—OBSTRUCTIVE HYPERTROPHIC PULMONARY EMPHYSEMA

	Air	Oxygen	Helium	After Epinephrin Inhalation
Rate	14	14	17	16
Ventilation cc./Min.	9,000	7,795	10,365	9,515
Oxygen Absorption cc./Min.	269	228	269	269
Vital Capacity cc.	2,280	2,985	2,800	2,780
Maximum Expiratory Velocity cc./Sec.	490	695	595	725
Maximum L./Min. Ventilation	15	14		

TABLE V.—LARYNGEAL OBSTRUCTION DUE TO CARCINOMA NECESSITATING TRACHEOTOMY

(See Fig. 4)

	Air	Oxygen	Helium
Rate	24	25	25
Ventilation cc./Min.	9,290	7,545	7,295
Oxygen Absorption cc./Min.	310	310	425
Vital Capacity cc.	2,156	—	2,146
Maximum Expiratory Velocity cc./Min.	501	—	754
Subjective Relief		0	+

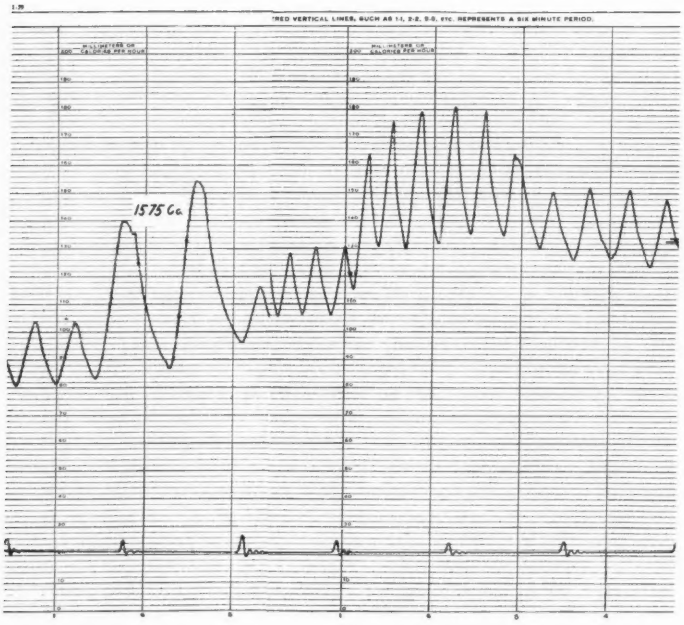

Fig. 3. Obstructive-pulmonary emphysema and bronchial asthma. Reduced vital capacity, 1,575 cc., with less curve on expiration than in asthma alone (Fig. 2) and absence of supplemental air. Maximum ventilation reduced to 18 L. per min. with air trapping.

Laryngeal and Tracheal Obstruction: Referring to Fig. 4, and Table V, the dyspnea of this type of obstruction generally shows an increased ventilation, moderate reduction in vital capacity and slowing of the velocity of air movement. The vital capacity curves at the inspiratory position are usually symmetrical in contradistinction to the other types of obstruction discussed above.

Oxygen is usually beneficial and helium more so as is evidenced by the reduced ventilation and increased air velocity.

Anoxia Test: Referring to Fig. 5, in a patient without anoxia, increasing the oxygen concentration causes no appreciable change in the rate of oxygen absorption. In the patient with anoxia due to pulmonary abnormality, the higher oxygen concentration causes a temporarily increased rate of oxygen absorption until the blood be-

comes saturated at the higher oxygen tension, and then the rate drops to its previous level, i.e., the oxygen deficit is satisfied. If the patient's breathing is regular so that accurate oxygen absorption rates can be determined, the test can be utilized although it is not very sensitive.

TREATMENT

The functional examination of respiration is of considerable value in the treatment of obstructive diseases. For this interpretation the examination consists of short periods of controlled and measured therapeutic trials thereby revealing responses which would be evident only after considerable periods of therapy. It is also possible to test rapidly and easily various procedures which otherwise would require rather elaborate apparatus and considerable time.

Routine testing generally consists of using oxygen concentrations of 50% to 90% helium in oxygen, positive pressure and epinephrin subcutaneously and by inhalation.[5]

As improvement occurs many patients become free of complaints; a further study of respiration will frequently reveal the persistence of the abnormal mechanism and a favorable response to the therapeutic procedure. This has been found especially true in bronchial asthma. After some improvement many of these patients have declined their epinephrin inhalations. The respiratory examination at this time has repeatedly shown persistence of the characteristic asthmatic curves and a favorable response to the inhalations. Under these circumstances continuation of the therapy regardless of the patient's subjective feeling of complete relief has proved to be of value (Table III).

It might be well to stress the accumulative nature of bronchial asthma, i.e., one attack predisposes to another, building up by retention of air in the chest to the continuous dyspnea or *status asthmaticus*. By the prevention of these attacks the frequency, duration and severity will be decreased.

RESEARCH

It has been demonstrated in medical science repeatedly that the quantitative determination of degrees of impairment of function is of value. The functional examination of respiration represents an effort to express more accurately the qualitative and quantitative

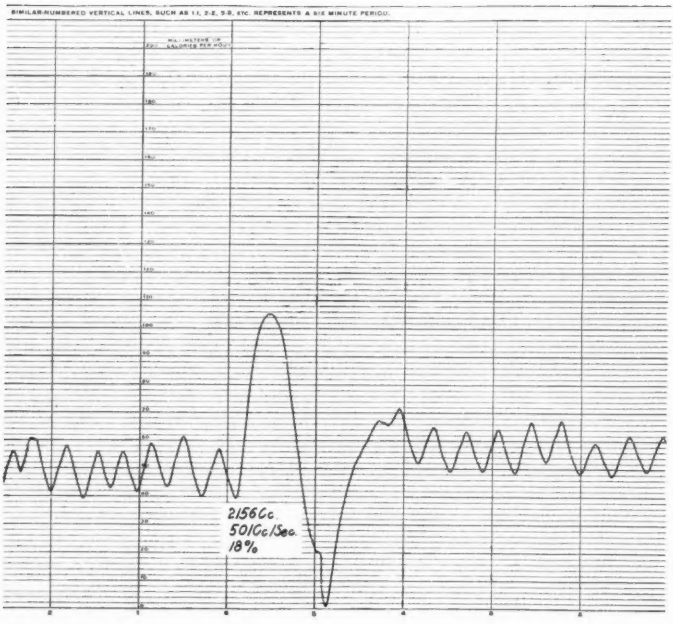

Fig. 4. Laryngeal obstruction due to carcinoma necessitating tracheotomy. See Table V. Reduced velocity of air movement, 501 cc. per sec. on expiration and symmetrical slowing of velocity at the extremes of the vital capacity especially at inspiration. Vital capacity, 2,156 cc.

alterations of respiration in abnormal conditions and thereby fills a gap in the study of respiratory disease.

A correlation between the bronchoscopic findings and the characteristics of respiration will, I believe, contribute to our knowledge of the pathological physiology and the treatment of certain obstructive diseases of the bronchi especially bronchial asthma and hypertrophic pulmonary emphysema.

At the present time three deductions may be made from our observations; these are as follows:

1. Positive pressure applied to the airways results subjectively in easier breathing in those patients who show marked collapse of the trachea or main bronchi on expiration.

2. The majority of patients with considerable quantities of thick secretion insufficiently removed by cough and ciliary action, show a variability in successive vital capacity determinations generally of an increasingly obstructive nature. It is this type of patient with bronchial asthma which is most consistently benefited by bronchoscopic therapy.[6]

3. Patients with laryngeal, tracheal and high bronchial obstruction show a symmetrical retardation of the rate of air flow at the extremes of the vital capacity determinations. (Patients with marked collapse during expiration are not included in this group.)

DISCUSSION

A functional examination of respiration can be made in spite of the discomfort associated with dyspnea. However, it is essential to have the apparatus in perfect condition and to obtain the cooperation of the patient, which usually is the easiest part of the examination. In the unconscious patient or in extreme dyspnea when the patient's entire effort is only respiratory, a face mask can be substituted for the usual mouth piece and nose clip.

The psychological effect on the patient is frequently beneficial inasmuch as he feels that his essential complaint has been tested and that the therapeutic procedures being used have been tested and found to be effective.

This examination does not in any way replace any other diagnostic procedure. It is an additional examination which accurately and graphically demonstrates the air movements which otherwise can be recognized only by inspection and auscultation of the chest. The measurement of vital capacity is only of limited value, but when the qualitative characteristics as well as the actual volume are considered, the significance is greatly increased.

The therapeutic procedures mentioned above are directed toward the relief of the dyspnea, but the fundamental causes for the obstruction must receive their deserved attention.

The majority of patients studied by this method have been severe bronchial asthmatics referred for bronchoscopic consideration. Therefore in an effort to classify the many factors involved, the following outline has been developed.

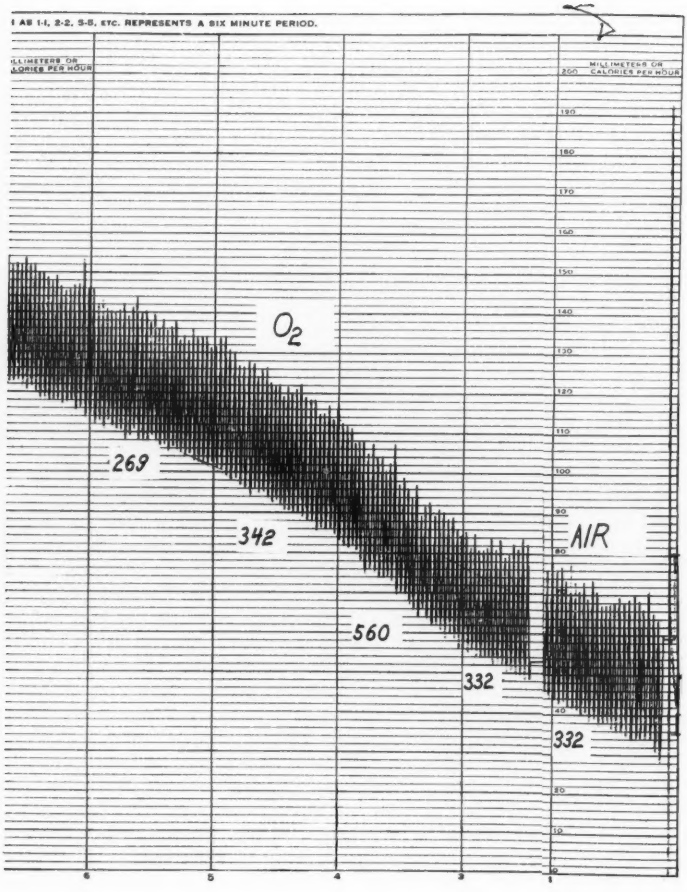

Fig. 5. Anoxia test, on patient with pulmonary edema following relief of a spontaneous pressure pneumothorax. Figures show the amount of oxygen absorbed per minute. Increased rate of oxygen absorption in the second and third minute of the test during which the oxygen desaturation of the blood was satisfied, thus indicating anoxia.

DIAGNOSIS OF BRONCHIAL ASTHMA*

Etiological:

Allergy
  Exogenous
  Endogenous
Associated disease at onset
Respiratory infection
Extra pulmonary disease.

Pathological:

Minimal-allergic bronchitis
Chronic bronchitis
  Catarrhal
  Suppurative
  Insufficient bronchial drainage
Bronchial collapse
Emphysema
Atelectasis.

Functional:

Respiratory embarrassment .
Respiratory decompensation—anoxia
Therapeutic response
  Oxygen
  Helium-oxygen
  Positive pressure
  Epinephrin.

*Adapted from the "Classification of Heart Disease" of the American Heart Association.

SUMMARY

A clinical method for the functional examination of respiration has been described which records the movement of air in and out of the chest by means of a modified basal metabolism apparatus. The normal findings and the findings in tracheal obstructions, bronchial asthma and obstructive emphysema have been described.

The diagnostic, therapeutic and scientific usefulness of the examination have been discussed, and an outline of the diagnosis of bronchial asthma has been presented.

25 E. WASHINGTON ST.

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LXX

PERFORATION OF THE TRACHEA BY A MEDIASTINAL TUMOR (HODGKIN'S DISEASE)*

ARTHUR E. HAMMOND, M.D.

DETROIT

During the past few years, the value of bronchoscopy in the diagnosis and treatment of the various diseases of the pulmonary tree has acquired an ever-increasing importance. Lately the method has gained still greater impetus by the addition of the X-ray laminagraphic procedure which permits the cross-section of the thorax and the structures contained therein into layers at successive depths, thus visualizing the particular lesion observed by the bronchoscope in much greater detail than was heretofore possible.

REPORT OF A CASE

A white male, aged 15 years, entered Harper Hospital as a medical staff patient on October 13, 1940. He gave a history of having been well until two months prior to admission when he suffered from attacks characterized by dyspnea, wheezy breathing, orthopnea, cough productive of thick yellowish non-foul smelling material, more pronounced in the evening, and slight generalized chest pain. Some relief had been obtained from adrenalin. Similar attacks had recurred at two-week intervals lasting from a few hours to several days. There had been a weight loss of 20 pounds during the last two months. The past history and family history were essentially negative. The physical findings revealed an anemic undernourished boy who appeared chronically ill. The chest was symmetrical with equal expansion. Breath sounds were quiet with slight purring on expiration. Rales and rhonchi were present in both lungs. No adenopathy was noted. Blood pressure was 110/70; T. 98.6; P. 96; R. 28. Bronchial asthma and pulmonary tuberculosis were considered in the tentative diagnosis. Roentgen study of the chest by Dr. Lawrence Reynolds revealed some increase in the width of the supracardiac mediastinum to the right side suggesting glandular enlargement.

*Presented at the 24th annual meeting of the American Broncho-Esophagological Association, Cleveland, Ohio, June 3, 1941.

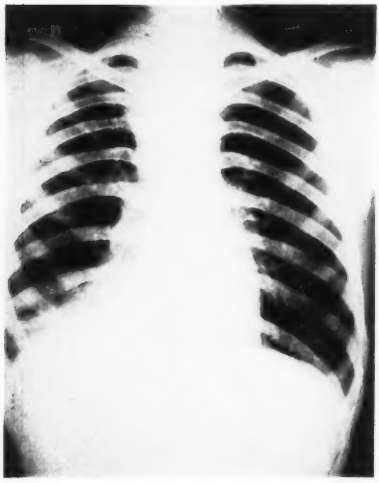

Fig. 1. Roentgenogram showing an acute parenchymal process involving the right middle and to a greater extent the right lower lobe.

There was also noted an acute parenchymal process involving the right middle and to a greater extent the right lower lobe (Fig 1).

Laboratory: The blood count on October 14, 1940, was: Hgb. 90; RBC 5,190,000; WBC 17,000; differential PMN 76%, L 15%, M 4%, E 5%.

Urinalysis—Negative.
Skin tests—Negative.
Blood Sugar—FO. 111. NCN. 35.2.
Blood Kahn—Negative.
Sputum—Negative for acid fast bacilli.

Clinical Course: The patient continued to have attacks of dyspnea and could not lie in the prone position. Bronchoscopic examination was considered at this time but was advised against because of his poor physical condition. Repeated sputum examinations were negative for the tubercle bacilli. All skin tests were negative. A check-up examination of the chest on October 27, 1940, revealed an increase in the width of the supracardiac mediastinal shadow to

the right side of the sternum with some displacement of the trachea to the left and the patchy parenchymal consolidation in the right middle and lower lobe areas still present. The cough had now taken on a brassy character as if the recurrent laryngeal nerve were being irritated. The temperature fluctuated irregularly between normal and 101 . The blood count on October 26th was: Hgb. 88; RBC 5,160,000; WBC 26,750; differential PMN 88%, L 10%, M 2%.

Sternal puncture on October 31st showed marked hypoplasia of white cell and erythroblastic tissue, also a marked increase in monocytes and eosinophils with myeloid elements showing toxic degenerative changes.

The opinion of Dr. William Tuttle of the thoracic surgery department at this time was that the patient was suffering from a lymphosarcoma or a malignant thymoma which was compressing the lower trachea and right main bronchus with resulting dyspnea and pneumonitis of the right lower lobe. Bronchoscopy was again deemed inadvisable because of the dyspneic state. Deep X-ray therapy in small doses was advised at once.

The patient was seen by the special tumor clinic on October 28th and was referred to Dr. T. Leucutia for deep X-ray therapy. At this time no other adenopathy was noted except a left axillary gland.

Following the first course of irradiation, the patient was markedly improved except for occasional attacks of nausea and vomiting. The dyspnea disappeared, the temperature became normal and there was very little sputum. He was discharged on November 10, 1940, with a diagnosis of mediastinal tumor.

After returning home he continued to improve and gained 25 pounds in weight. He returned as an out-patient for further deep X-ray therapy on December 9, 1940 and January 6, 1941. About February 1, he again developed considerable cough with the return of his former obstructive symptoms. Further irradiation was given on February 17th and 18th. The following day the dyspnea was quite marked, and he again entered the hospital on the medical service. The positive physical findings were labored breathing with an expiratory wheeze, with rhonchi and rales throughout the chest. Small glands were noted in the axillary and inguinal regions. The temperature was 99, P. 90, R. 24, B.P. 125 80. With rest in bed the dyspnea gradually disappeared, and the X-ray examination of the chest again showed a definite improvement. At this time the patient

Fig. 2.

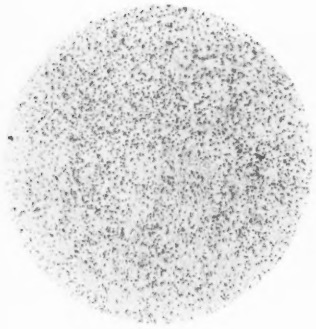

Fig. 3.

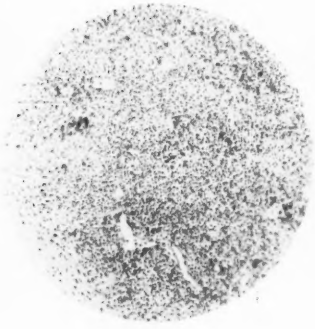

Fig. 4.

Fig. 5.

Fig. 2. Chronic granuloma-like material showing a diffuse fibrosis with round cell infiltration and proliferating fibroblasts.

Fig. 3. Showing marked hyalinization of the connective tissue.

Fig. 4. Photomicrograph showing giant cells of the Dorothy Reed type.

Fig. 5. High power view of the same specimen as that shown in Fig. 4.

Fig. 6. Laminagraph showing mediastinal tumor projecting through tracheal wall into the tracheal lumen. Film made by Dr. Lawrence Reynolds.

was referred regarding the advisability of a bronchoscopic examination. In reviewing the history and roentgen findings, I felt that a diagnostic bronchoscopy was indicated, even though the evidence to date pointed to an extratracheal tumor and that the bronchoscopic findings would be only indirectly confirmative. With the present improved physical condition and no apparent respiratory embarrassment, bronchoscopy appeared to be a relatively safe procedure and was so advised.

Bronchoscopic Examination, March 4th: Under local anesthesia, a 9 mm. bronchoscope was introduced. The larynx and upper trachea were negative. The lower tracheal lumen was partially obstructed by a tumor mass attached to the right tracheal wall just above the carina. A portion of this tumor was pedunculated and moved back and forth with respiration into the right main bronchus. The bronchoscope was slowly passed by the lesion to the orifice of the right upper lobe bronchus which was open and not invaded by the tumor. The lower border of the tumor mass was about 1 cm. above the right upper lobe bronchus. A large portion of this tumor

mass was removed with biopsy forceps for microscopic study. My impression was that of a tuberculoma or a malignancy.

A second bronchoscopy was done on March 6th to remove more tissue for microscopic study and to enlarge the airway as much as possible. A decided improvement in breathing followed.

Pathology: The report of the microscopic examination of the tissue by Dr. Plinn F. Morse was as follows: First biopsy: specimen consists of tissue detritus and necrotic granulations containing a few cells of highly atypical appearance. A presumption of some form of neoplasm is strongly suggested by the appearance of these cells. Second biopsy: tissue consists of a mass of chronic granuloma-like material which upon further study reveals a diffuse fibrosis with round cell infiltration and proliferating fibroblasts. In the older areas the connective tissue has become hyaline and is quite typical of the later lesions of chronic Hodgkin's (Figs. 2 and 3). In other areas the process is more active and giant cells of the Dorothy Reed type along with an occasional eosinophil are made out (Fig. 4). Under higher power the character of the granulation is quite typical of active Hodgkin's disease (Fig. 5).

Following the bronchoscopic examinations, another X-ray study was made at this time with the laminagraphic method in order to visualize, if possible, the encroachment of the tumor upon the tracheal lumen. The films were exposed at 3, 4 and 5 cm. depths, the best detail of the trachea being obtained with the 4 and 5 cm. depths. A tumor extended into the lumen of the trachea from the level of the episternal notch to the bifurcation of the trachea on the right side with narrowing of the lumen of the trachea to about one-half normal width in transverse diameter. This tumor lay directly opposite the shadow of the tumor mass, which extended from the mediastinum to the right side of the sternum projecting into the right lung field just below the level of the episternal notch (Fig. 6).

The case was again presented before the tumor committee on March 10, 1941, and it was decided to sustain the diagnosis of Hodgkin's disease and to administer further treatment. On March 17th and 18th, the fifth deep X-ray therapy series was given. Since the bronchoscopic removal of a greater portion of the intratracheal tumor, there were no further attacks of dyspnea or wheezing, and the breathing was much improved. Attacks of nausea and vomiting continued to recur, however, and the patient was discharged on March 19th following the final X-ray therapy treatment.

The patient continued to be nauseated and vomited frequently at home. There were no further attacks of dyspnea. With the persistence of the vomiting, he pursued a rapidly downhill course and expired at home on April 28, 1941.

CONCLUSIONS

1. A case of a mediastinal tumor (Hodgkin's disease) with perforation into the trachea is presented.

2. The value of bronchoscopy for the establishment of diagnosis and outline of therapeutic procedure is emphasized.

3. The contribution of laminagraphic study of the tracheobronchial tree and mediastinum in the diagnosis of endo- or extra-tracheal or bronchial tumors is noted.

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LXXI

PRESENTATION OF THREE INTERESTING ENDOSCOPIC FOREIGN BODY CASES*

MAURICE BONNIER, M.D.

MONTREAL

Three unusual cases of foreign body, two in the air passages and one in the esophagus and stomach, are presented to emphasize certain points of clinical interest and to discuss the methods of treatment.

REPORT OF CASES

CASE 1.—*Tracheal Cannula in Bronchus.* The patient, a man, aged 36 years, had a tracheotomy performed on May 30, 1939 for obstruction due to carcinoma of the larynx. Three weeks after operation the tubular portion of the No. 5 cannula became detached from the shield and was lost in the trachea. On August 24, 1939 an unsuccessful attempt elsewhere at removal was made under general anesthesia. When admitted to the clinic eight days later the patient exhibited no signs of respiratory distress. There was moderate fever.

Physical examination of the chest revealed harsh breath sounds over the right chest with moist rales over the lower one-half of the lung. There was no evidence of bronchial obstruction. Roentgen studies of the chest revealed a large crescentic shadow resembling a curved cannula with the lower extremity in the right bronchus and the upper end resting on the carina (Fig. 1).

Bronchoscopic removal was performed four days after admission. A 9 mm. x 30 cm. bronchoscope was employed. The upper end of the cannula was secured with forward grasping forceps and was anchored in the tube so that during traction a rotating motion from left to right could be performed until the fistula was reached; then traction was directed anteriorly and downward as one does when removing a tracheotomy tube. There was no reaction following removal.

*Presented at the 24th annual meeting of the American Broncho-Esophagological Association, Cleveland, Ohio, June 3, 1941.

A *B*

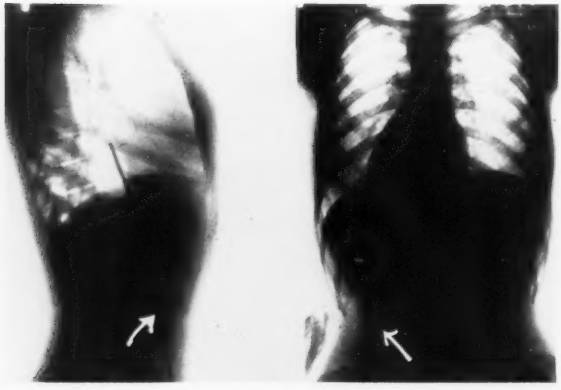
Fig. 1. Roentgenographs, *A*, showing a tubular portion of large tracheotomy tube lodged in great part in right bronchus with uppermost extremity resting on the carina; *B*, lateral view showing the portion above the carina.

The foreign body was 9 cm. in length and 10 mm. in its greatest diameter. The lumen being unobstructed allowed air to pass freely in and out of the right lung and therefore the degree of obstruction was insufficient to produce atelectasis. The lodgement of the foreign body across the carina with an end in each main bronchus would unquestionably have produced more serious results.

The patient died two months later from the effects of metastasis of the carcinoma to the kidneys.

CASE 2.—*Corset Bone in the Esophagus and Stomach.* While standing on a chair to pull down a window, the patient, a woman aged 21 years, slipped and by some unexplainable means swallowed the foreign body. There was immediate discomfort behind the sternum. The following day this was referred to the espigastrium. Attempts to take food produced abdominal pain and a sensation that something was bending below the ribs.

Roentgen examination revealed a long thin opaque object resting vertically in the axis of the esophagus. The lower end was observed in the fundus of the stomach and the upper extremity in the esophagus (Fig. 2).

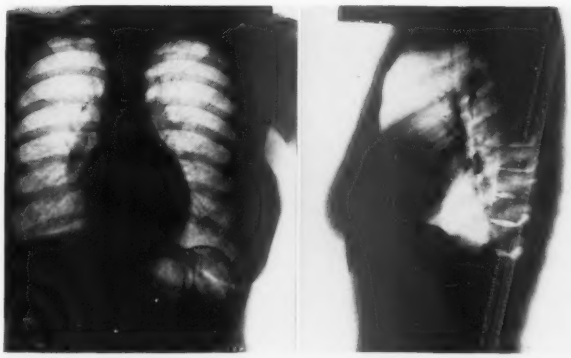


A B

Fig. 2. The long foreign body shadow is readily observed in the lateral view *(A)* and extends from the lower thoracic esophagus to the greater curvature of the stomach corresponding to a point slightly above the level of the pelvic brim. In the roentgenogram *(B)* the upper end is hidden by the heart shadow; the lower end indicated by the arrow is in the fundus of the stomach.

With a 9 mm. x 45 cm. esophagoscope the object was promptly localized and easily removed. It was 21.5 cm. in length and 6 mm. in diameter. The ends were rounded and smooth and the shaft was uniformly pliable. This probably explains the absence of any evidence of injury to the pharynx or esophagus.

CASE 3.—*Safety Pin in Bronchus.* The patient, a woman, aged 41 years, was referred to the clinic for removal of a safety pin from the right bronchus. She had previously consulted her physician because of fever, pain in the right chest and slight hemoptysis of two weeks' duration. The safety pin was discovered in a routine roentgen study of the chest. The patient then recalled that she had accidentally swallowed a safety pin ten years previously, but the absence of symptoms other than slight choking and cough at the time of the accident suggested that the pin had been expelled in a stool. On careful questioning it was discovered that during the intervening years there were episodes of fever, cough and sputum and chest signs suggesting bronchitis or bronchopneumonia. She had been advised



A *B*

Fig. 3. In the roentgenogram *(A)* the open safety pin is observed in the plane of the right lower lobe bronchus. There is remarkably little roentgen evidence of change in the lung distal to the foreign body. In the lateral view *(B)* the point and keeper are directed upward, the point being anterior.

to rest as pulmonary tuberculosis had been suspected, but at no time was a roentgen study of the chest suggested nor was a foreign body considered as a possibility by her physicians.

On admission her general condition appeared satisfactory. Examination of the lungs was negative except for moist rales below the level of the right hilar region. Roentgen study revealed a small open safety pin in the lower lobe bronchus of the right lung. The point and keeper were directed upward, the point being anterior and slightly outward (Fig. 3).

Bronchoscopic removal with the aid of biplane fluoroscopy was carried out under avertin anesthesia. This form of anesthesia was employed as the patient was allergic to drugs used for local anesthesia. An attempt was made to employ the straightening technic of removal, but the rusted pin broke at the spring when pressure at the lip of the tube was exerted. It was interesting to note that the suppurative process was slight and bleeding scanty despite the granulations observed on an ulcerated area of the anterior wall of the bronchus where the point of the pin had been imbedded. Two

months following bronchoscopy the patient expectorated two small hard, brownish calculi. These appeared to be deposits of calcium which were detached from the corroded pin at the time of removal; it is possible that these were deposited in the bronchial wall at the site of ulceration in contact with the point

This case demonstrates the extraordinary adaptation of a secondary bronchus to an expanded, pointed, ulcerating metallic foreign body over a period of ten years. The point only was imbedded firmly in a thickened ulcerated granulating wall; the remainder of the foreign body was free in the bronchus. Seven weeks following removal a complete examination of the chest was negative.

Without roentgen study, which was undertaken because of hemoptysis, the presence of a safety pin probably never would have been suspected.

410 Sherbrooke St., W.

LXXII

MEDIASTINAL ENLARGEMENT IN ACUTE LARYNGOTRACHEOBRONCHITIS*

G. W. Grier, M.D.

Pittsburgh

For a number of years cases of acute tracheobronchitis have come to my attention which show an enlargement of the superior mediastinal shadow on X-ray films of the chest. Because of this enlargement persistent thymus is usually suspected, and the question of carrying out radiation treatment has to be decided.

Of course there is no reason why a baby with persistent thymus may not contract tracheobronchitis also, but the condition I wish to describe is not due to thymus and I believe can usually be differentiated from it without applying the therapeutic test of radiation.

I have been in the habit of calling this condition mediastinitis on the theory that it is due to a swelling and inflammation of the structures in this location, probably accompanied by a slight enlargement of the paratracheal lymphatics. This would seem to be borne out by the fact that the few cases which we have been able to follow up with X-ray films later have shown a decrease in the size of the mediastinal shadow. We have not had an opportunity to follow up very many cases, and the interval before re-examination has varied considerably. Two cases showed a distinct decrease in size in a period of about a week, two other cases at the end of a month, and four cases at varying intervals up to a year. Two cases which were re-examined showed no change in size.

There have been only three autopsies on our patients who have died during an acute attack, and in none of these did the pathologist report any abnormality noted in the mediastinum. However, it is only fair to state that the pathologist was not aware of these unusual X-ray findings and no special effort was made to look for any unusual pathology which might produce them. Only one of these cases showed a widened mediastinal shadow on the X-ray film.

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*Presented at the 24th annual meeting of the American Broncho-Esophagological Association, Cleveland, Ohio, June 3, 1941.

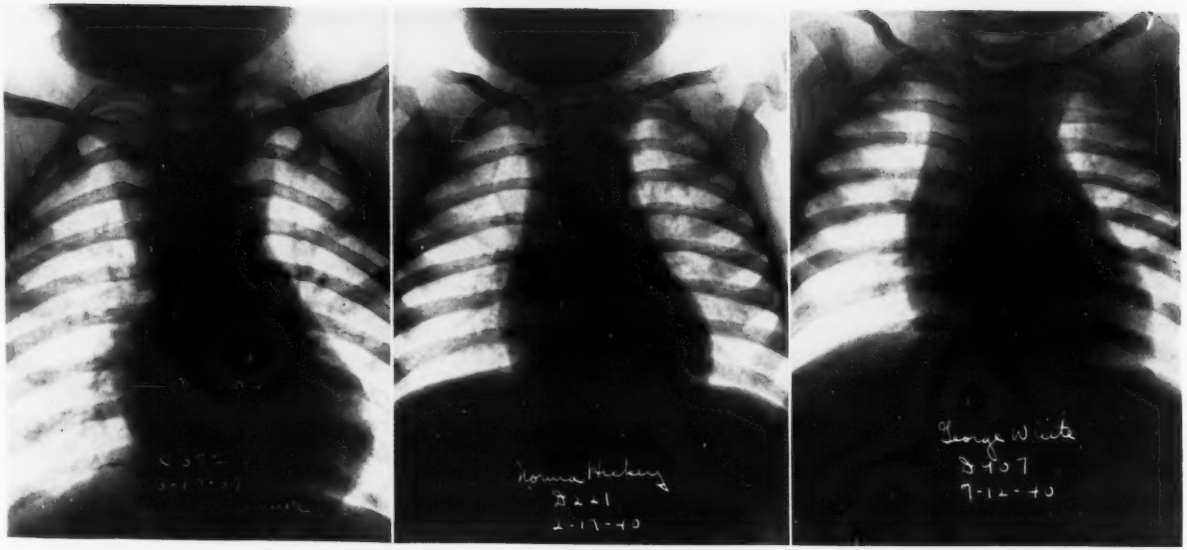

Fig. 1. Three cases of laryngotracheobronchitis, all of which show the typical biconcave shadow as described in the text.

The shape of the mediastinal shadow is rather characteristic, being a symmetrical shadow which projects on both sides of the spine, and the edges are usually concave, but occasionally straight.

In contradistinction the thymus shadow is almost always a bulging shadow, the borders being convex, and it is not uncommon for it to project to one side of the spine only.

The shadow cast by enlarged lymphatic glands is also usually a bulging shadow with a greater tendency to be irregular or nodular in appearance and usually on one side only.

The shadow of a new growth such as lymphosarcoma or Hodgkin's disease is also apt to be larger and bulging in character.

Mediastinal abscess, usually a sequel of acute infection which itself might be indistinguishable from tracheobronchitis, also presents a smooth convex border quite different from that described above.

There are, of course, exceptions to these general rules, but the majority of cases conform to these descriptions sufficiently that a differentiation can usually be made.

Of these mediastinal enlargements the one most apt to present an appearance similar to that seen in tracheobronchitis is persistent thymus. There are, of course, symptoms common to both, and as a matter of fact a baby with persistent thymus may develop acute tracheobronchitis.

We have had seven such cases, four of which were treated by radium and recovered, one recovered with symptomatic treatment and two died before radiation treatment could be carried out. Of the two that died, autopsy was performed on one, and both tracheobronchitis and enlarged thymus were demonstrated.

With this experience in mind, a baby with tracheobronchitis presenting a mediastinal shadow which could be interpreted as persistent thymus should have the benefit of the doubt and be treated by radiation.

An especially confusing picture is presented by infants with congenital heart lesions who contract tracheobronchitis. The shadow of the great vessels is quite apt to be enlarged and distorted in these lesions, and it is often difficult to decide whether the abnormal mediastinal shadow is a part of the heart condition or the result of the tracheobronchitis.

The discussion of the differentiation of congenital heart lesions is too complicated and too lengthy to consider in this article, but a

few fundamental facts may be mentioned. Naturally dyspnea and cyanosis are common to both.

The abnormal shadow of the heart stem seen in congenital heart lesions may look quite like the widened mediastinal shadow seen in tracheobronchitis.

In the latter condition, however, there is no particular reason for an abnormally shaped heart, while in congenital heart lesions the enlarged great vessels are, with very few exceptions, accompanied by alterations in the normal shape of the heart.

A child may have both tracheobronchitis and a congenital heart lesion. We have recognized four such cases in our series.

Shortly after we first noticed the widened mediastinal shadow in cases of tracheobronchitis, the same thing was noticed in bronchopneumonia.

In our service at the Children's Hospital, cases of pneumonia are much more common than those of acute laryngotracheobronchitis, and since pneumonia is readily recognized on the X-ray film and tracheobronchitis is not, for a long time we had the impression that the sign of widened mediastinal shadow indicated pneumonia.

In this article I have not attempted to tabulate cases of pneumonia not accompanied by tracheobronchitis which show widened mediastinum on the X-ray film, but I am sure they are more numerous than the cases of tracheobronchitis.

The percentage of positives to the total number of cases in pneumonia may not be as high as in tracheobronchitis, but the total number seen I am sure is greater.

CASES OF LARYNGOTRACHEOBRONCHITIS SEEN AT CHILDREN'S HOSPITAL, PITTSBURGH, BETWEEN JAN. 1, 1938 AND MAY 1, 1941

Without enlarged mediastinum	8
Plus pneumonia without enlarged mediastinum	5
With enlarged mediastinum	20
Plus pneumonia with enlarged mediastinum	14
Plus enlarged thymus	7
Plus congenital heart lesion	4
Plus tuberculosis, enlarged mediastinum	1
Total	59

Since January 1, 1938, 129 cases of laryngotracheobronchitis, or tracheobronchitis, have been diagnosed at the Children's Hospital, Pittsburgh. Of this number, 59 have had X-ray films of the chest. The accompanying table analyzes these cases.

Only 13 out of 59 cases presented a normal mediastinal shadow, while 34 cases showed the typical widened shadow that I have attempted to describe.

The fact that autopsy failed to reveal any marked enlargement of glands or other abnormal masses should not be accepted as proof that no pathology has existed *in vivo*. Swelling and edema could conceivably disappear at postmortem.

An interesting observation is the presence of this sign in a patient who had an open safety pin in the esophagus for two or three days previous to the X-ray examination. When the esophagoscope was passed on this child it stopped breathing every time the scope reached this area. Fortunately the pin went on down into the stomach ahead of the esophagoscope and was passed per rectum in due time.

This case was seen only two weeks ago, so sufficient time has not yet elapsed to know whether or not the widened mediastinal shadow will disappear spontaneously.

It seems probable that any infection in the region of the mediastinum, which, of course, would include the trachea and esophagus, or in the lungs, particularly the upper lobes, may produce this sign.

I do not believe that enlarged lymphatic glands *per se* play very much part in the abnormal X-ray shadow, although the small number of cases we have had come to autopsy (three) is hardly conclusive on this point.

500 Penn Ave.

Society Proceedings

JOINT MEETING

OF THE

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

AND THE

MILWAUKEE OTO-OPHTHALMIC SOCIETY

March 11, 1941

DR. J. B. HITZ, PRESIDENT OF THE MILWAUKEE SOCIETY, IN THE CHAIR

Cavernous Hemangioma of Nose, Nasal Septum and Forehead

SAMUEL SALINGER, M.D.

(Abstract of Case Report)

This patient, a girl aged 20, was first seen in May 1937 for relief of an oversized nose which had been present since birth. There was a smooth enlargement of the lower half of the nose, of bluish-purple color, elastic to the touch and extending almost to the level of the lower border of the nasal bones. The lower portion of the septum bulged into the nares, causing almost complete obstruction. The mucous membrane was very thin and pulsated slightly. The entire mass was easily compressible externally and internally. To the left of the midline of the forehead was a similar mass extending from the hair line toward the supraorbital margin. While the color was much like that of the nasal swelling the mass felt somewhat more dense.

It was felt that surgery was contraindicated and the patient was referred to the Michael Reese Tumor Clinic where interstitial radiation was applied. Inasmuch as the septal portion of the growth remained practically unchanged, although there was reduction in the size of the nose, it was decided to treat it more directly by implanta-

tion of radium needles. Following this, it was apparent in November 1939 that the vascularity of the masses had been greatly reduced, and plastic procedures were undertaken for cosmetic purposes.

In January, 1940, the mass on the forehead was excised in toto with the overlying skin and the defect closed by undermining the adjacent skin, which was brought together with tension sutures. This mass extended to the periosteum. The bulbous nose was reduced by two parallel excisions of elliptical wedges of skin and underlying stroma on each side of the nose from the level of the lower border of the nasal bones to within a few millimeters of the alar margins. The resulting scars were not entirely satisfactory and were resutured a short time later with much better cosmetic effect. A few pit marks will be excised at a future time.

Histologic examination of the tissue showed a large amount of fatty tissue irregularly distributed throughout the stroma, some muscle tissue and a large number of blood vessels showing various degrees of hyalinization. The vessel changes were undoubtedly due to the application of radium.

The case is reported because of its comparative rarity and to demonstrate the value of preliminary irradiation for reduction of the vascularity of these otherwise inoperable tumors. The essayist felt that this was a case of developmental anomaly.

DISCUSSION

Dr. Harry Foerster (Milwaukee): I wish to compliment Dr. Salinger on obtaining a very excellent cosmetic result in a difficult case. The case he presented is unique in some respects, as indicated by the histopathology. It represents a developmental anomaly rather than a new growth. I recall a case Dr. Mulsow and I had at the county dispensary some years ago, in which the patient had numerous vascular lesions involving both the skin and mucous membranes and in which the pathologist reported the tissue removed as lipoma. The lesions were vascular tumors with an abundance of proliferative fatty tissue. Cavernous angiomas that are in the midline, in the location of this patient's lesion, may be due to developmental defects involving the deeper tissues and even extending to the meninges. This is something to bear in mind when employing surgery on highly vascularized lesions in similar locations.

The surgical procedure is to be discussed by the next speaker, who has had much experience in this line. The advisability of not

attempting to rely on one procedure, but of using a combination of methods, should be emphasized. There is no question that some angiomatous lesions can be satisfactorily removed surgically, and also that many other cases are amenable to radiotherapy alone, and the cosmetic results may be very good. There are, however, certain cases in which the results with combined treatment, as cited in this case report, are far more satisfactory than by either method alone.

Dr. Salinger spoke of pits in the skin which had to be corrected. This is an important point in connection with interstitial irradiation. Unless the physician is experienced he is likely to produce localized areas of atrophy in the skin because of the radium needles not having been planted deeply enough. As he said, implants of radon seeds are sometimes preferable because of this, but a difficulty with radons is that the seeds may move around in the loose vascular tissue after they have been planted, and if they get too close to one another, necrosis may occur, while in other parts of the field, from which the seed has moved, inadequate radiation effects necessitate further treatment.

The most satisfactory results in the treatment of cavernous angiomas are obtained with irradiation in those cases which are treated in infancy or early childhood. As the patient becomes older the angiomatous tissue becomes more mature and it is then less radiosensitive, and therefore to obtain an effect one must depend on destruction of tissue by the radiation. In such cases an alternative treatment that has proved very satisfactory and that is to be recommended is the injection of sclerosing solution, of which the most satisfactory is probably quinine hydrochloride and urethane. This method likewise requires a certain amount of experience but not nearly so much as radium therapy, nor is there the same risk of necrosis. Permanent damage of the skin by irradiation is not infrequent, whereas the injection of sclerosing solution, if carried out with proper technic, may produce very satisfactory results.

The remarks with reference to the ineffectiveness of superficial radium therapy in cases like the one discussed, should be stressed by being repeated. Such lesions are not amenable to surface applications of radium because the dosage required to destroy the angioma is bound to damage the overlying skin irreparably.

Dr. George V. I. Brown (Milwaukee): Dr. Salinger's interesting illustrations of the good results he has secured by combined treatment and operation are worthy of commendation, because in reconstructing the disfigured nose he has accomplished the necessary

improvement by the use of immediately surrounding tissue. This I feel is a very important matter, because so often flaps are transposed from more or less distant parts, and although the appearance of a nose so constructed may be satisfactory so far as outline is concerned, the tissue almost always continues to look as though it belongs somewhere else and not on the face.

In the case of one infant an angioma covered almost the entire side of the face as well as the nose, the lips and the lower eyelid, and seemed to be almost hopeless from an operative point of view. Yet it recovered spontaneously. In a second case there was an angioma on the side of an infant's head and face, which grew to enormous size in 18 days. After ligation of both external carotids without benefit, the child finally came for operation, the end result of which was effective. Such angiomas may continue without outward change from birth and in middle life begin to grow rapidly and require immediate operation.

The face and eyelids of one infant were covered by a pigmented hairy mole. Most of the forehead and the upper part of the face was cured by skin grafting. Part of the growth was left for an eyebrow. There were nine of these pigmented moles on this infant's body, and in the course of a few years all have disappeared spontaneously except one which is evidently disappearing. In contradistinction to this, there are similar cases where the pigmented surface has been excised and the whole skin graft placed, yet there is noticeable a slight tendency to recurrence of the pigment through the transplanted skin.

The purpose of discussing these cases that cured themselves without treatment and those which without apparent difference in macroscopic appearance or microscopic examination grew rapidly, is to bring out the little understood differences in the reaction, which probably explains the contradictory reports of the results of treatment by various methods.

DR. SAMUEL SALINGER (closing): I would like to say that I appreciate the discussion, and also want to emphasize a point that Dr. Foerster mentioned, namely, that these congenital blood tumors are more successfully treated in infancy and early childhood than when the patient reaches adult life. Also, there are so many different types of these tumors about the face, occurring at different levels and of different degrees of vascularity, that the treatment must necessarily vary according to the location, size and variety of the lesion. We therefore find different agents being used in different

cases. Surgery, surface irradiation, interstitial irradiation, electrodessication and carbon dioxide snow are all successful if intelligently applied. In many cases it will require the combined judgment of the surgeon, dermatologist and radiologist to determine what is best for the individual case.

Early Diagnosis of Carcinoma of the Esophagus

J. R. LINDSAY, M.D.

(This paper is printed in full on p. 675 of this issue.)

DISCUSSION

DR. JOHN GORDON (Milwaukee): Those of us who have had to do with malignant diseases of the esophagus know what the prognosis has been up to the present time. When we make a diagnosis of malignant disease of the esophagus, we have to prepare ourselves to sign the death warrant for the individual.

With the marked development of thoracic surgery in the past ten years there seems to be much more to offer these individuals provided they can be seen in the early stages of the disease. Unfortunately, most of us see these cases far too late. The thing that sends them to us is not the early symptoms but the late signs, such as difficulty in swallowing soft foods or even inability to swallow fluids. Usually by this time there has been such a degree of extension to the surrounding parts that surgical removal is almost impossible.

If we as a medical group are to help further the work of the thoracic surgeon in removing these neoplasms, it will have to be done through a broadspread educational campaign to the laity and to the attending physicians, so that the individual with esophageal symptoms will be seen at a very early stage.

Esophagoscopy offers one of the methods of examination which many times will permit of a definite diagnosis far in advance of X-ray or fluoroscopy, and when there is any question as to lesion or diagnosis an esophageal examination should be made.

The men who are doing the newer types of esophageal resection for malignant disease deserve a great deal of credit. Up to the present time they have had little reward for their efforts. I believe that with earlier diagnosis and earlier resections the mortality of esophageal malignancy cases will be reduced.

Report of a Case of Chronic Suppurative Otitis Media, Facial Paralysis, Circumscribed Meningitis and Acute Labyrinthitis

HOWARD C. BALLENGER, M.D.

(This paper was printed in full in the ANNALS, 50:286 (March), 1941.)

DISCUSSION

DR. T. L. TOLAN: I think it is encouraging to know, in these times of the sulfonamides, that patients such as Dr. Ballenger described got well even before the new therapy. It seems impossible at this time to feel that such patients would get well unless we could give sulfanilamide credit for the cure. I think it is particularly interesting that the patient had a seventh nerve paralysis before operation and developed a sixth nerve paralysis after the operation.

The case is interesting, and I think Dr. Ballenger is to be congratulated in pursuing a watchful waiting attitude, after thorough exenteration of the mastoid.

DR. ALFRED LEWY: In Dr. Ballenger's case there was no positive bacterial culture of the spinal fluid, indicating that it was not a generalized meningitis but probably secondary to the local infection. My diagnosis would be that it was a chronic mastoiditis with petrositis probably, and the other intracranial symptoms, paralysis of the seventh and sixth nerves, were due to edema of the brain. I have a similar case in the hospital now. Before sulfanilamide we usually had recovery from the meningitis with negative cultures but not in the case of positive cultures.

The patient I have is a negro boy who should have been operated on last December when we first saw him. He came in a few days ago with a fullblown meningitis. There was still some hearing left in the ear, and we did the usual radical operation with uncovering of the dura in both middle and posterior fossae. The dura was changed in color, not thickened and not under pressure. The day after operation the boy had complete aphasia and complete hemiplegia on the right side. The neurologist's idea was that there was a sphenotemporal lobe abscess. My idea was that it could not have developed in so short a time. On the second day the boy recovered movement of the right arm and leg but was still aphasic. He is now recovering his speech. I hope he recovers. I do not think he had an abscess, but the neurologist insists that he did.

DR. SAMUEL SALINGER: Dr. Ballenger brought up a point on which I would like to have him elaborate, namely, the question as

to whether the labyrinthitis was secondary to the meningitis or the meningitis secondary to the labyrinthitis. This has been mentioned in the literature and was definitely proven by Ruttin in one case where an autopsy showed that the labyrinthitis was due to retrograde infection through the internal auditory canal from meningitis. In certain cases the indications for treatment might depend on having this information. I would like to have Dr. Ballenger give his reasons for his conclusions.

Dr. Howard Ballenger (closing): I want to thank Dr. Tolan for his discussion. I am glad to have Dr. Lewy's analysis of this case; however, a petrositis with an edema of the brain would not account for the spinal fluid cell count of 1,100. Some intracranial infection was present. Roentgen rays showed nothing other than chronic mastoiditis. So far as I know there was no evidence of petrositis.

In reply to Dr. Salinger's question, the symptoms of meningitis were present before the patient was admitted to the hospital. The symptoms of labyrinthitis developed later. For that reason I assume if an infection did occur in the labyrinth it was by way of the internal auditory meatus.

Kodachrome Motion Picture Studies of Pathology of the Larynx

Paul Holinger, M.D.

The color motion picture presented illustrated various types of laryngeal pathology as seen through the direct laryngoscope. Acute and chronic inflammatory lesions and laryngeal neoplasms were illustrated. The author's equipment for laryngeal photography was described.

DISCUSSION

Dr. Herbert Schmidt (Milwaukee): These moving pictures of the larynx speak for themselves. They are so beautifully done that it appears to be a simple matter, but to take such pictures requires a high degree of skill in photography. Certainly we will all agree that this is an ideal way of showing conditions in the larynx, even the normal larynx. For teaching purposes it will be invaluable in demonstrating pathology of the larynx. In one evening a student or a doctor may see lesions or conditions which could otherwise be

seen only over a long period of time if he had to wait until such cases could be observed in the clinic.

I presume in the future Dr. Holinger will be able to photograph many more interesting cases that come to his attention, and will probably show us more and more of these laryngeal conditions.

Dr. Walter Theobald: I would like to comment on the necessity of the type of work that is being done by Dr. Holinger at the bronchoscopic clinic at the University of Illinois, at St. Luke's Hospital and at Children's Memorial Hospital. The importance of direct laryngoscopy became apparent to me in the case of a man who had been hoarse for six months. On one side there was a very marked bulging red infiltration of the larynx beginning at the right false cord and extending onto the true cord, and which evidently was subglottic. Also, there was a slight deviation of the larynx and trachea at this point to the left side, as shown by X-ray. With this long history of hoarseness I could think only of malignancy. To make a very thorough and exhaustive study, and to be sure it was not malignant, we recommended examination by Dr. Holinger. We found the entire infiltration to be perfectly smooth. We did find, however, a growth extending toward the trachea from the thyroid gland. I feel sure we are not dealing with a malignancy but perhaps a thyroid growth outside the trachea. The basal metabolic rate shows a range of +50 to +29. The patient is now on potassium iodide and at a later date will probably have thyroid surgery. The importance of being sure we are not dealing with a malignancy is shown by this case, and we can only be sure through direct examination.

Dr. John Mulsow: I have often thought that if it were possible, it would be interesting in this type of photography if one could insert the lateral X-ray picture to show the amount of tumor invasion of surrounding tissue, not seen in direct or indirect laryngoscopy. I do not know whether it could be done, but it would be interesting.

With reference to Dr. Theobald's case, some time ago I saw a patient who had considerable difficulty in breathing, with evidence of pressure on the trachea, and the X-ray showed a large substernal goiter.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, April 7, 1941

THE PRESIDENT, DR. ROBERT H. GOOD, IN THE CHAIR

Surgical Anatomy and Technic of the King Operation for Bilateral Abductor Paralysis of the Vocal Cords*

LINDON SEED, M.D.

(Abstract)

The essayist discussed the operation presented by King in 1939, and expressed the belief that this is by far the most satisfactory procedure thus far devised for this condition. Most patients heretofore have been subjected to permanent tracheotomy. This paper is designed primarily to illustrate the anatomic landmarks and to describe the technic. The slides which accompanied the presentation were drawn from dissections of fresh and preserved cadavers and from freshly removed larynges, and showed the technic of operation in detail.

The operation is basically an arthrodesis of the arytenoid joint with the arytenoid fixed in abduction and external rotation. Although the procedure is not formidable, it must be carried out meticulously. King has admonished that no one should undertake the operation without first acquiring an accurate knowledge of the anatomy involved, and suggests that such knowledge be acquired by actual work in an anatomic laboratory.

DISCUSSION

DR. E. SASSON: The patient shown presents one of the rather rare conditions of Riedel's struma. She came with a complaint of

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*From the Department of Surgery, College of Medicine, University of Illinois, and Cook County Postgraduate School, Chicago.

dysphonia and choking at meals. Six months before, her neck had begun to swell. Two weeks later she entered with urgent dyspnea requiring tracheotomy. The left cord was fixed and pushed toward the right. The whole larynx was infiltrated; the glottic chink was very narrow. There was a smooth, very hard swelling over the thyroid gland, which was slightly tender and fixed to the surrounding deep structures but not to the skin. Regional lymph nodes did not seem involved.

Two histologic types of chronic thyroiditis are described: (1) Riedel's struma, which shows much fibrosis with considerable hyaline degeneration; (2) Hashimoto's "struma lymphonatonis" which shows marked infiltration with lymphocytes. There has been considerable controversy concerning their relationship. The slides demonstrated from this case show both types in the same case, which suggests that there is no essential difference in the underlying pathology.

The patient has had fractionated X-ray therapy to a total of 4,800 R., and now has a fair voice and normal airway. Although considerable thyroid swelling is still present, she has had no trouble without a tracheotomy tube for several months and seems to be nearly recovered.

Modified Kelly Operation for Bilateral Abductor Paralysis

T. C. GALLOWAY, M.D.

(Abstract)

Since postoperative bilateral abductor paralysis is such a distressing condition, it is fortunate that at last real progress has been made in its treatment. The Hoover and Love operations were fairly satisfactory, and the procedure of King is a real advance. Kelly's idea of an approach through the thyroid cartilage seems to simplify the technic greatly.

The patient I presented had the King operation 17 months ago on the right side and a modified Kelly operation on the left side seven months ago, and offers a good comparison of the two. During the King operation under local anesthesia the omohyoid muscle was seen to contract on deep inspiration. For several weeks afterward the arytenoid to which it was attached did the same, but there was no movement on ordinary breathing, though the position was fair.

After a few months the condition was only a little better than before operation.

On the other side the Kelly approach was used with two modifications:

1. A direct laryngoscope was inserted under the epiglottis, and then under vision through the window in the thyroid cartilage, the arytenoid was easily picked up by a sharp hook. This seemed important, as its position, as recently pointed out, may vary considerably especially in the two sexes, and it may not be too easy to locate.

2. The arytenoid was left in, as it seemed that its attachment to the cord through the vocal process made much better traction possible lateralward.

The operation was done (1) through a Kocher's incision; (2) the perichondrium elevated; (3) a round window made with curet and biting forceps; (4) the arytenoid grasped by a hook through its muscular process guided by direct vision through a laryngoscope; (5) the cricoarytenoid joint mobilized; (6) the cricoid transfixed anteromedial to its center of rotation, drawn laterally into the space of the thyroid cartilage window and fixed to the thyrohyoid muscle and external perichondrium. The result seems excellent with a fair voice and a quite adequate airway, and the cord is holding as it was after operation.

Dr. Seed's beautiful demonstration of the mechanics of arytenoid movements seems important. Probably a good many operative failures have been due to disregard of point of fixation through the arytenoid.

A tracheotomy should always be done when the patients have much dyspnea to prevent cardiac collapse under prolonged strain or acute emergency, as well as in preparation for later operative relief.

DISCUSSION

DR. WARREN COLE: I really am not qualified to discuss this paper unless it would be because I had followed King's work very closely and am convinced there is real merit in his operation. With Dr. Holinger I operated on one patient which, however, was a failure, as I shall mention later.

I do not believe that transplantation of the omohyoid is an important part of the operation, and the observations of Galloway of which he told us convince me that it is not important. Dr. Seed has

illustrated the important point that the suture must be properly placed; if placed far posteriorly on the muscular attachment the pull on the fulcrum will be wrong, and widening of the space between the cords will be minimal, and if this is so I would expect it to decrease as time passes and perhaps be insufficient.

Of the two procedures I think the King operation is superior to the Kelly. The one advantage of the Kelly operation is that you remove some substance, the arytenoid cartilage, thereby making room; the consequent atrophy of muscles would also tend to increase the size of the airway. But in the Kelly operation there is danger of perforation through the mucosa resulting in infection and perhaps abscess formation. I think Dr. Galloway's procedure is a splendid contribution and I believe it will be used, at least in part. The great advantage is that it is quite simple, and with the window cut in the thyroid cartilage you do not have to stand on your head to get exposure of the cartilage. However, it seems likely that there would be fewer landmarks and one might have difficulty in getting one's bearings, particularly since the arytenoids may not always be in a constant position, as brought out previously by Dr. Lederer.

The patient on whom Dr. Holinger and I operated was about 65 years of age, and had had paralysis of the cords for seven or eight years; she came in with complete respiratory block. At operation we obtained a fairly good exposure of the cartilage and disarticulated it satisfactorily. The cartilage was extremely brittle and fractured when the suture was placed. We replaced the suture and after it was tied laryngoscopic examination revealed a good airway. Something happened later to reverse the whole picture, because a few hours later she had almost a complete block. I am quite certain that the suture either broke or pulled out, perhaps because of another fracture of the cartilage. I would therefore like to issue a caution about elderly patients. You must be careful about putting the suture in. In King's publication he also told about breaking two or three cartilages. Naturally, the tendency toward calcification will be greater in elderly people. It might be advisable to dissect the cartilage loose so that a suture may be placed around rather than through it.

Dr. Alfred Lewy: Perhaps I should apologize for passing an opinion on an operation I have never seen. There has recently come to my attention an operation by Krainz, of Innsbruck, for bilateral abductor paralysis with median position of the cords. His operation is done under suspension and consists essentially in submucous eleva-

tion of the anterior and posterior mucosal walls of the interarytenoid region and the removal of the *transversus arytenoideus* muscle with a Patterson punch. Krainz reported four cases at the 1939 meeting of the German Otolaryngological Society. All these patients were wearing a trachea tube with which they were able to dispense after the operation. He claims they also had a very good voice. This he attributes to the tensor action of the cricothyroid muscle, which is supplied by the superior laryngeal nerve and not involved in the recurrent nerve paralysis. In complete bilateral recurrent nerve paralysis the cords are in the cadaveric position. The median position he believes is due to contracture of the arytenoideus muscle. I mention this operation because it is simpler than any that has been advocated so far. I have had no opportunity to perform it myself.

In one case of bilateral paralysis with median position of the cords in a school teacher who was faced with the dilemma of choking to death or losing her job and starving to death if she lost her voice, I put in a Gordon New check valve trachea tube, which enables her both to breathe and to speak so well that she has been working for over a year without her colleagues discovering that she is wearing the tube. She refused operation for a long time until an upper respiratory infection brought about an acute obstruction that required temporary intubation.

DR. LINTON SEED (closing): I had a discussion with King about a month ago. Although in his original paper he emphasized the attachment of the omohyoid to the muscular process of the arytenoid, in his second paper he said definitely that the vocal cord to which is attached the omohyoid does not move at all. It is absolutely fixed in all the patients he has seen. In an autopsy on a patient who died later, in whom the transposition of the omohyoid had been performed, he noticed that there was some widening of the vocal slit produced by pulling on the transposed omohyoid.

I do not believe that Dr. Galloway has modified the Kelly operation but has modified the King operation. What he has done is to rotate the vocal process of the arytenoid cartilage in an outward direction and hold it there with string. Dr. Galloway's approach through the thyroid plate seems more simple and I believe can be performed more easily. Dr. King himself found that in some cases he could facilitate the operative procedure by taking a lateral bite out of the thyroid plate, thus giving more room.

As to the benefit to be derived in the simple severance of the interarytenoideus muscles, I can only say that in the cadaver the

severance of these muscles produces no widening of the vocal slit. Widening depends almost entirely upon rotation of the arytenoid cartilage.

The interarytenoideus muscles are paralyzed in a recurrent laryngeal paralysis, and they should not be causing more adduction. However, if four such operations have been done with successful results, one cannot lightly cast it aside, although theoretically it may seem relatively inefficient.

Scarlet Fever, Bilateral Mastoiditis With Intervening Pneumonia, Cerebellar Abscess and Recovery

ROBERT B. LEWY, M.D.

(Abstract of Case Report)

The patient was a boy aged four years, who was admitted to the Municipal Contagious Disease Hospital with the diagnosis of scarlet fever. The long and stormy course from August 26, 1940, to January 11, 1941, was given in detail, and the author discussed the complications which arose and the measures used in combating each.

The conduct of the case was of particular interest because all along the line important decisions had to be made; not infrequently there was disagreement among the clinicians as to what should be done, when it should be done and how it should be done.

The first decision to be made was: since mastoidectomy was definitely indicated on the left ear, and the right ear had discharged less than two-thirds as long, should surgery be performed on this as well. It was finally decided to operate on the left ear and to judge by the findings there whether the right ear should be operated on. The pathology found was adequate to justify operation on the left mastoid but did not indicate that the inflamed mastoid bone on the right side was adequately necrosed and localized for surgery.

The second decision to be faced was to establish the cause of the continued temperature after the first operation. There were two possibilities, either or both of which could have been the cause: pneumonia, and the continued right mastoiditis.

The third decision was whether the right mastoid was responsible for continuance of the pneumonic process and should be operated on or, as the pediatrician held, whether surgery on the mastoid would further lower the patient's resistance. Finally a right mastoidectmy under local anesthesia was performed, with a favorable result until development of a left cerebellar abscess.

The fourth decision concerned when to operate on the cerebellar abscess. Because of the comatose condition of the patient surgery was performed the same day that diagnosis was established.

The fifth decision involved the recommendation of the neurologist, who felt that trephining should be performed on the side opposite the abscess, draining the ventricle and suboccipital approach made into the abscess. Instead of this, the mastoid opening was enlarged to uncover the dura over the posterior fossa and the abscess was drained directly from this area.

The sixth decision was an easy one and concerned the use of sulfanilamide after diagnosis of cerebellar abscess was made. The reasoning here was simple. It was felt there was everything to gain and nothing to lose. Since in the characteristic pathology of the abscess there is a very vascular wall, large quantities of sulfanilamide would deposit in this region and might prevent progress while nature had a chance to get in and heal; 140 gr. were given per day with this idea in view.

DISCUSSION

DR. SAMUEL SALINGER: It is certainly nice to hear about a brain abscess that recovered, and congratulations are due Dr. Lewy and his consultants on the happy result. I often wonder, when I see or read about these cases, who has the most headache, the patient or the doctor, because I have been through this sort of thing and know all about the discussions and arguments that take place.

Dr. Lewy poses a number of questions which I believe merit some comment. I can agree with him as to the logic of his procedures with one or two exceptions. The first point that I think debatable pertains to the question of doing a bilateral mastoidectomy in a scarlet fever patient with both ears discharging. I am in favor of waiting until I can be reasonably sure that the process is progressing beyond the point where it is not likely that resolution will take place spontaneously. I much prefer to do the two sides at the same time in order to see the degree of pathology and to be sure that the foci

have been eliminated. Dr. Lewy stated that he was going to judge by the degree of pathology present on one side as to how far the process had progressed on the other. I do not believe this is a safe criterion. In the first place the ears began to dischage at different periods, indicating different degrees of local and constitutional resistance, and it is just as likely that the second ear might have progressed more rapidly than the first, since it became involved at a time when the patient's resistance was lower than in the beginning. In the second place, without knowing definitely the degree of pathology present in the ear not operated upon, he was forced into the dilemma of having to decide whether the etiology lay in that ear or beyond the surgical field of the side operated upon. The decision would have been much easier had both mastoids been opened at the same time.

As to the question of opening the brain abscess through a clean area or approaching it by way of the original field, I am afraid that the subject is still controversial. For my own part I can say that the best results I have seen have come from opening the abscess through the original operative field.

Concerning the use of sulfanilamide after the abscess had been opened, I agree that no harm could come from its use in the hope of combatting bacterial activity in the capsule of the abscess and the adjacent vascular bed. However, I do think that the effectiveness of the remedy would be greatly enhanced by using it locally as well as internally. General surgeons have found the local use of sulfonic compounds extremely valuable in the treatment of large infected spaces and advocate it by irrigation, instillation of heavy suspensions or packing with the pure powder. It seems to me that if it is safe to use in the peritoneal cavity it should be equally safe in a brain abscess.

Dr. Archibald L. Hoyne: At the hospital we felt that this was a very remarkable piece of work that Dr. Lewy accomplished. Concerning the use of sulfanilamide, we are not particularly enthusiastic in regard to its value for scarlet fever. We have been using it locally for some patients who have had aural discharge, and we have been giving it orally sometimes to see whether our results were as favorable as many claim. There has been a great deal of doubt as to its benefit. Some of us thought it was very helpful while others did not.

There is one point that has not been emphasized particularly, that is, the time when the complications developed in Dr. Lewy's

patient. The child was in the hospital with what might be considered as an average case of scarlet fever. He was progressing very favorably. At the beginning of the fourth week, first one ear and then the other developed trouble. This is frequently so in mild attacks of scarlet fever. After the patient has practically recovered, complications may come on which can terminate fatally. The ear complications of scarlet fever are often serious. I think Dr. Lewy is to be congratulated on his excellent management of the case reported.

DR. L. J. HUGHES: Did this child have sulfanilamide prior to the onset of the ear complications and, if so, do you think it had something to do with deferring the otitis media?

DR. ROBERT B. LEWY (closing): This child had no sulfanilamide prior to the time the ears began to discharge, but did have some after the ears began to discharge. That was because I did not come in on the case or it would not have been given. I feel that it masks the picture, and we have enough intangible questions in these cases.

With reference to Dr. Salinger's discussion, I think it is interesting to look at the history of mastoidectomy in this country in the last 35 years. Many of you will remember that mastoids were operated on in the hemorrhagic stage, and that is still recommended in some textbooks. You will also recollect that 25 or 30 years ago there was a far higher incidence of intracranial complications than there are today. I cannot say for sure that these tie in together, but it might be that the performance of the mastoid operation before there was ample time for localization was the reason many intracranial complications started.

This is one case that might argue against that. Nevertheless I believe there is a distinct co-relationship. I always hesitate before I operate on any mastoid area unless there are danger signs. In this case we were in a spot where one mastoid had gone so far we felt it was dangerous, and we felt it was hazardous to go into the other. For that reason we operated on one at a time.

About the use of sulfanilamide by irrigation: I have read about its being used in compound fractures, but I have not been convinced that it was the sulfanilamide rather than good care that brought about the results. If sulfanilamide is used for irrigation it comes up against a surface, whereas if it is in the blood stream it will prevent further infection.

MAX A. GOLDSTEIN

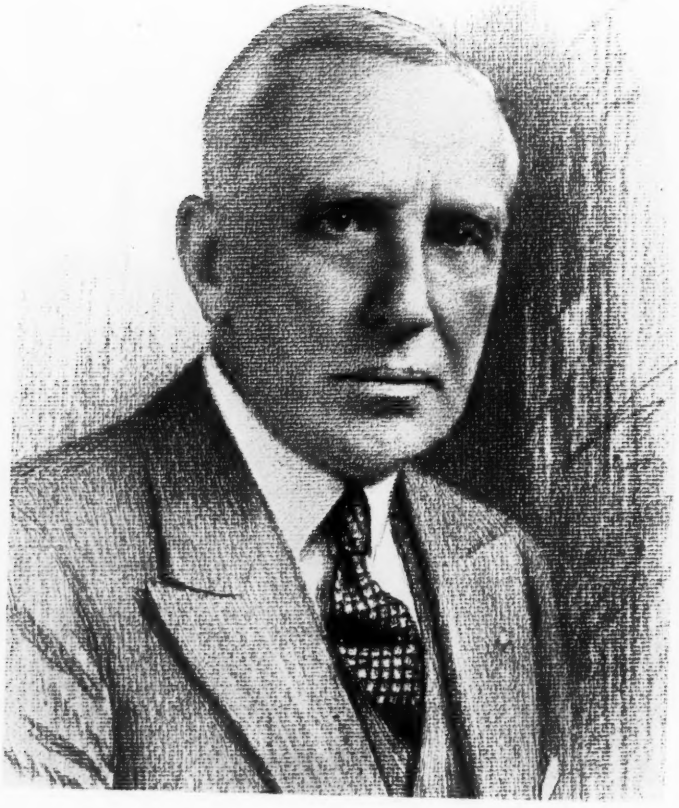
1870-1941

Dr. Max A. Goldstein of St. Louis died at his summer home in Frankfort, Michigan, on July 27, 1941 at the age of 71 years.

A native of St. Louis, Doctor Goldstein received his preparatory training in the public schools of that city and his medical degree from the Missouri Medical College. Following a year's interneship in the St. Louis City Hospital he repaired to Vienna for special training in otolaryngology under Adam Politzer and Victor Urbantschitsch. After further study in Berlin, Strasbourg and London he returned to St. Louis and began the practice of otolaryngology in 1896. Almost at once he undertook the publication of the Laryngoscope, which he owned and edited until the time of his death.

His absorbing interest, however, was always the education of the deaf and especially the deaf child. This culminated in 1914 in the foundation of the Central Institute for the Deaf, which has since assumed a position among the first in the country. He was a strong advocate of the oral method of training, even in the days when the sign language was the method of communication accepted by the deaf themselves and by their teachers. Doctor Goldstein busied himself not only with the development of this method of teaching but also with the endowment of his Institution, which owing largely to his efforts has often been the recipient of large donations. Graduates and teachers trained in this institution now number several hundred.

In recognition of his services the city of St. Louis presented him in 1933 with its Annual Award for Distinguished Service. In 1937 Washington University conferred upon him the honorary degree of LL.D. Doctor Goldstein was a fellow of all the national laryngological societies and the president of the American Otological Society in 1927 and of the American Laryngological, Rhinological and Otological Society in 1931. He was the founder of the Society of Progressive Oral Advocates, which later became the National Forum on Deafness and Speech Pathology, and its president for 25 years preceding his death. He founded also the St. Louis League for the Hard-of-Hearing, was a director of the American Federation of Leagues for the Hard-of-Hearing and of the American Society to Promote Teaching of Speech to the Deaf. From 1937 to 1939 he was president of the American Speech Correction Association.



Max A. Goldstein

He served during the World War as a major in the Medical Corps and as chief of head surgery at Camp Dodge, Iowa.

His contributions to the literature dealt mainly with the rehabilitation of the deaf. These were epitomized recently in two books, "Problems of the Deaf" and "An Acoustic Method for Training the Deaf and Hard-of-Hearing Child."

Mrs. Goldstein, the former Miss Lenore Weiner, and a daughter survive him.

Doctor Goldstein made many friends in and out of the profession who are now engaged in collecting an endowment for the Central Institute in his memory.

Books Received

Applied Physiology of the Nose.

By Arthur W. Proetz, A.B., M.D., Professor of Clinical Otolaryngology, Washington University School of Medicine. Cloth, 8vo, xxii + 395 pages with 91 illustrations. Annals Publishing Co., St. Louis, 1941.

Of all the basic sciences in medicine physiology is considered one of the most important. In recent years the difficult problem of the management of certain diseases such as diabetes and pernicious anemia, for example, has been solved on the basis of a more complete understanding of the organs involved.

Now Dr. Proetz presents a complete and comprehensive volume on the applied physiology of the nose. Many of the old theories of function have been disproved and many new principles have evolved from more recent contributions. A large proportion of the material consists of the author's own observations and original research experiments. His observations on the ciliary activity and its relation to physical and chemical agents are most noteworthy. After reading this text one is convinced of the importance of taking into consideration the physiology as well as the pathology in the local and surgical treatment of the diseases of the nose and paranasal sinuses.

This monograph represents an outstanding contribution to rhinology. It should prove enlightening to every student and practitioner of otolaryngology who is interested in keeping pace with the latest trends in this field of medicine.

F. K. H.

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